# PHOTIC STIMULATION AND THE TREATMENT OF MOOD AND SLEEP DISORDERS.

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January, 2004

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## DECLARATION

I declare that this thesis reports my original work, that no part has been previously accepted and presented for the award of any degree or diploma from any university, and that, to the best of my knowledge, no material previously published or written by any other person is included, except where due acknowledgement is given.

Kerry Ellen Leahan

## ACKNOWLEDGEMENTS

I wish to thank the following people for contributing, each in their own way, to the completion of this thesis:

Professor Don Byrne, who believed in me and steadfastly encouraged and guided me in the process of research, and never accepted anything but the best. A special thank you for support given, during desperate times, to acquire the specialised equipment and technical support needed for this research.

Dr. Mike Smithson, for his statistical support and professional guidance.

Dr. Cobie Brinkman, for the many hours of guidance and proof reading, and for helping me to improve my writing skills.

Professor Robert Gregson for his interest in this research, and for trying so hard to steer me in the direction of non-linear dynamics.

Mr Graham Kelly, and the technical staff at the ANU, for their support, and readiness to help.

Professor Barry Sterman, for generously supplying the Mindset, without which my research would not have been finished, and for teaching me so much about electroencephalography and how to stick doggedly to the experimental process.

Dr Consuelo Barreda-Hanson for her support and generosity in making space available in the Psychology Department at The Canberra Hospital for me to conduct my second study, and for tolerating my juggling of work and study commitments, all of which has enabled me to bring this thesis to a close.

Dr. David Noton for his very generous supply of the 'Lightmasks' for use in this research.

Mr. Terry Eichmann for his ongoing support and assistance with processing many of the EEG files, and the invaluable sharing of his technical and EEG expertise.

Mr. Steve Parkes for his ongoing interest and support and for generously sharing his knowledge with me.

Ms. Carmel O'Sullivan for her interest, and for making this thesis possible by assisting with the acquisition of the 'Lightmasks', which were used in the final study.

My friends who have offered support and encouragement over the years, I look forward to catching up with all the missed dinners, and to fun times ahead.

A very special acknowledgement, and thank you, to my family; Lorne, Jessica and Rebecca, who have weathered much, and yet have continued to offer their love, support and encouragement through the many years of study, and for knowing I had more strength and endurance than I realised myself. Without Lorne's technical skill and software expertise I would still be back at Study 1, laboriously extracting data.

Finally, to all those men and women who gave their valuable gift of time to participate in my research and who taught me so much.

# ABSTRACT

Major Depressive Disorder (MDD) is a stress-related disorder characterised by over-activation of the hypo-thalamic pituitary axis (HPA) (Arborelius & Owens, 1999; Nofzinger et al., 2000). Dysregulation of this system causes disturbances in behaviour, mood, cognition, and diurnal and circadian regulation of appetite and sleep-wake patterns. Abnormal electroencephalography (EEG) is also a common feature, with increased left midfrontal alpha indicating underactivity of the left frontal cortex relative to the right (Henriques & Davidson, 1991; Tomarken & Keener, 1998). Photic stimulation (PS) elicits cortical photic driving responses which can be used to 'entrain' the EEG, and produce rhythms conducive to relaxation and sleep onset, or conversely, to activate and 'switch on' the cortex in readiness for cognitive work. In addition, photic stimulation produces reliable anxiolytic effects, which can be used to reduce hyperarousal, and assist in the re-establishment of homeostatic balance, with the potential to be effective in the treatment of stress related disorders such as MDD.

Two studies explored the clinical utility of PS as a therapeutic tool in the treatment of mood and sleep disorder. Study 1 compared audiovisual stimulation (AVS) at 5Hz, 13Hz, and 22Hz to autogenic relaxation in a non-clinical sample of thirty, in a repeated measures (12 sessions), single blind, controlled design. AVS produced immediate relaxation responses as effectively as autogenic relaxation, with reductions in heart rate and skin conductance during stimulation, but with no evidence of long-term training effects on physiological arousal or EEG. Main effects for mood (PANAS) were found. Negative affect decreased accompanied by unexpected decreases in positive affect over sessions. Interaction effects were small and non-significant with trends showing enhanced positive affect and subjective relaxation with 13Hz AVS, while AVS at 22Hz tended to increase arousal. Depression and anxiety symptoms were significantly decreased (SCL-90-R), even in a non-clinical sample.

Study 2 used a double blind, placebo controlled, repeated measures design. Sixty participants with MDD and accompanying insomnia received PS of 5Hz, 13Hz,

22Hz, or continuous light, while a wait list group received no treatment. Participants used a Lightmask™ at home for 20 minutes each evening over a period of 28 days while maintaining sleep and mood diaries. Measures of EEG, mood (PANAS) and depression (BDI-II) were taken at baseline, following 2 weeks of PS, after 4 weeks of PS, and at follow-up, 2 months post light therapy. Anxiety (SCL-90-R) was assessed at baseline and follow-up only.

Entrainment responses were observed in the majority of participants but were unrelated to mood. Davidson's pattern of alpha asymmetry in depression was found with increased left midfrontal alpha, relative to the right. Main effects were prominent, with improvements in depression, anxiety, and symptom severity, plus increases in positive affect and decreases in negative affect over time. These improvements were independent of group membership. Similarly, sleep efficiency improved, night wakings decreased, and waking mood increased, for all groups over time, but with no change in sleep onset latencies. Five hertz PS appeared to offer slightly greater benefit for sleep disturbance.

This research extends and contributes to current knowledge on the clinical application of PS, with the use of placebo controlled, random allocation, double-blind procedures. Results showed that PS induces immediate relaxation responses that contribute to treatment outcomes. Entrainment effects, however, did not enhance treatment outcomes. Strong placebo and non-specific therapeutic effects were present in both studies as both placebo and control groups showed improvements similar to active PS. In conclusion, PS elicits immediate relaxation responses, which according to Benson (1975) are essential in the treatment of stress-related disorders.

# CHAPTER 1

## 'Stress, EEG, and photic stimulation'.

### 1.1 Introduction

Stress is a part of everyday life. Selye (1976) defined stress as "the nonspecific response of the body to any demand" (p. 55). What he was describing was a generalized physiological reaction to stressors in the environment or arising from within the organism, caused by some internal cue. When confronted with threat or challenge the resulting 'stress' response includes an immediate 'alarm reaction' or generalized arousal caused by sympathetic nervous system excitation; heart and respiratory rate increase, blood pressure rises, the pupils dilate, the sweat glands are activated, blood glucose levels rise, blood is redirected from digestive functions to peripheral muscles of action, which gives rise to the feeling of 'butterflies' in the stomach, we may feel apprehensive, fearful or agitated, and even become hyper-vigilant. These responses are highly adaptive if the organism needs to flee from threat or face it and fight.

The stress response itself is fairly universal, but what we perceive as 'stressful' is not. Lazarus and Folkman (1984) refined Selye's definition of stress saying that stress is dependent on our 'appraisal' of the situation. Primary appraisal involves assessing whether or not a stressor is in fact a threat to well-being and therefore warrants action or 'coping'. Next, in secondary appraisal, we appraise whether or not we have the coping resources available to deal with the perceived stressor. Adaptation occurs when we successfully manage to deal with the stressor (Lazarus, 1991a; Lazarus & Folkman, 1984). If we don't manage to adapt and reduce the threat posed by the stressor, the stress response continues.

When stress is persistent and becomes chronic, according to Selye, during the 'stage of resistance', the organism adapts to the heightened level of 'stress', but not without a cost to the organism. There may be mood and sleep disturbances, loss of appetite, diminished libido, and compromised immune response, to name just a few. Persistence of stressors, both environmental and psychological, and their concomitant physiological stress reactions, can eventually lead to exhaustion, which is the end stage of Selye's "General Adaptation Syndrome". The relaxation response helps to counter the effects of chronic stress and restore the body to optimum functioning (Benson, 1975, 1985; Everly & Benson, 1989).

Herbert Benson (1975) defined relaxation as a generalised reduction in physiological arousal characterised by a decrease in heart rate, respiratory rate, oxygen consumption, muscle tension, blood pressure, and enhancement of brainwave activity within the alpha bandwidth. He reviewed many relaxation and meditation techniques and found that relaxation responses were induced by a few simple requirements: a quiet place free from external distractions, a repetitive word or phrase on which to focus in order to distract the mind, the adoption of a 'passive' attitude, and finally, a comfortable position that will facilitate the release of muscle tension. Traditional relaxation methods such as progressive muscle relaxation (Bernstein & Borkovec, 1973); autogenic relaxation, breath counting, yoga stretches, meditation (Arambula, Peper, Kawakami, & Gibney, 2001; Banquet, 1973; Fenwick et al., 1977; Kwon, Hahm, & Rhi, 1996; Orme-Johnson, 1973; West, 1980), biofeedback techniques using electromyography, temperature, or skin conductance, (Schwartz, 1987), and more recently, EEG biofeedback (Abarbanel, 1995; Banquet, 1973; Bird, Newton,

Sheer, & Ford, 1978; Lubar, 1989; Moore, 2000; Rozelle & Budzynski, 1995; Sterman, 1986, 1998; Sterman & Friar, 1972; Sterman, Macdonald, & Stone, 1974) and respiratory sinus arrhythmia training (Gevirtz, 1999; Lehrer, Vaschillo, & Vaschillo, 2000) meet some or all of Benson's criteria for induction of relaxation.

Relaxation techniques aim to re-establish homeostasis by reducing sympathetic nervous system input while enhancing parasympathetic nervous system activation in an attempt to override arousal mechanisms which cause stress and strain on the system (Benson, 1975, 1985). Many of these techniques, however, require a substantial investment of time and effort before the skills are acquired and the beneficial effects of relaxation can be realised. In our busy industrialised communities, there is an increasing demand for quick and easy relaxation therapies that will enable us to manage the stressors in our lives.

Mind machines or brainwave synchronisers are increasingly popular, require no learning, and claim to induce states of profound calm, or of heightened arousal, quickly and effortlessly. In accordance with Benson's prescription for relaxation, they provide repetitive stimuli, such as light and sound pulses, which distract the mind from its constant stream of thoughts and images. With the use of headphones and eyeglasses, they isolate the individual from their environment to create a 'quiet' personal space free from the influence of external input. Through the process of 'brainwave entrainment', certain frequencies of light and sound pulses act to 'pull' the brain into a relaxed state by creating brainwave frequencies which are normally associated with relaxation or altered states of consciousness with resultant reductions in muscle tension and enhanced well-being as the parasympathetic nervous system

takes predominance (Dieter & Weinstein, 1995; Shealy et al., 1990). Therefore, entrainment of the human electroencephalogram (EEG) can be achieved using photic or auditory stimulation that entices the EEG to respond in synchrony with the frequency of stimulation (Davis, 1939; Toman, 1941; Wada, Nanbu, Jiang, Koshino, & Hashimoto, 1996; Wada, Nanbu, Kadoshima et al., 1996; Wada, Takizawa, & Yamaguchi, 1995; Wada, Takizawaw, Kitazawa, Zheng-Yan, & Yamaguchi, 1994). Depending on the frequency of stimulation used different states of arousal and consciousness can be induced, as the human EEG tends to reflect different states of arousal and mood.

## 1.2 Human electroencephalography

The human EEG is a complex combination of synchronous and desynchronous cortical electrical activity with a wide spectral range. Early in his research, Berger (1929) noted that the different oscillations of the human brain were associated with different states of activation or arousal. With the help of techniques such as Fast Fourier Transformations, which today can be done effortlessly with computer software, the seemingly chaotic nature of the EEG could be decomposed into its various bandwidths. While there is no universal system for defining EEG bands, there is a general consensus that different EEG bandwidths are associated with different states of consciousness, mood, or levels of cortical activation.

Low frequencies within the delta bandwidth (0-4Hz) denote a cortex with minimal neuronal activity and consequently these are found during deep sleep, during coma, or seen in focal cortical areas following cerebral injury (Niedermeyer, 1993; Schaul, 1998). Theta (4-7Hz) activity is the dominant frequency found in children and

diminishes with age as the brain matures, although normal adult levels of theta may not be present in the EEG until age 25-30 years (Niedermeyer, 1993). Theta is associated with drowsiness (Lafrance & Dumont, 2000; Mulholland, 1995), increases during meditative states (Aftanas & Golocheikine, 2002; Kwon et al., 1996; Schacter, 1977; West, 1980), and is a necessary precursor to sleep onset (Lafrance & Dumont, 2000; Lafrance, Paquet, & Dumont, 2002; Strijkstra, Beersma, Drayer, Halbesma, & Daan, 2003). Theta usually signifies cortical deactivation, and its presence during task engagement can indicate problems with normal cortical functioning (Linden, Habib, & Radojevic, 1996; Lubar, 1991; Lubar & Lubar, 1984; Monastra et al., 1999; Ohashi, 1994). Conversely, recent evidence shows the presence of synchronised theta during the encoding process of memory performance, but with high theta power at rest being predictive of poor memory performance (Klimesch, 1999). In one study, low theta power, sometimes accompanied by an excess of beta power, was associated with psychiatric illness such as unipolar depression, bipolar disorder, alcohol and substance abuse, and mental disorders due to general medical condition (Coutin-Churchman et al., 2003).

The alpha rhythm (8-13Hz) is the dominant EEG frequency present in occipital leads when resting quietly with eyes closed (Basar, Schürmann, Basar-Eroglu, & Karakas, 1997; Berger, 1976 reprint of 1934; Hammond, 2002; Niedermeyer, 1993, 1997). The majority of healthy adults show attenuation of peak occipital alpha on opening the eyes, when stimulating the visual cortex, or during cognitive work (Basar et al., 1997; Micheloyannis, Papanikolaou, Bizas, Stam, & Simos, 2002; Neuper & Pfurtscheller, 2001; Niedermeyer, 1993, 1997; Williamson, Kaufman, Lu, Wang, & Karron, 1997). In exception, recent evidence in memory research shows

synchronisation of the upper alpha bands during encoding (Klimesch, Doppelmayr, Schwaiger, Auinger, & Winkler, 1999).

Alpha is a synchronous sinusoidal rhythm usually associated with a state of deactivation or 'rest', is often seen between periods of task engagement (Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990; Sterman, Mann, Kaiser, & Suyenobu, 1994; Williamson et al., 1997) and is associated with relaxation (Banquet, 1973; Marshall & Bentler, 1976; Morse, Martin, Merrick, & Dubin, 1977; Shimokochi, 1996; West, 1980) and positive mood states (Brown, 1970, 1971; Nowlis & Kamiya, 1970). When alpha is present during relaxation it is characterised by subjective feelings of 'relaxed alertness' in anticipatory readiness for the next cognitive task, rather than a state of relaxed drowsiness (Basar et al., 1997; Klimesch, 1999; Klimesch, Doppelmayr, Pachinger, & Ripper, 1997; Pfurtscheller, Stancak, & Neuper, 1996). The majority of healthy individuals have dominant alpha frequencies close to the 10Hz bandwidth (Basar et al., 1997; Boudrot, 1972; Hammond, 2002; Jabbari, Russo, & Russo, 2000; Niedermeyer, 1993, 1997; Toman, 1941; Walter, 1953), but individuals with higher resting alpha frequencies (>10Hz) have been found to perform better on memory tasks (Klimesch, 1999; Klimesch et al., 1997; Vogt, Klimesch, & Doppelmayr, 1998). In other research, photic stimulation at 10Hz, which enhances EEG power in the alpha range, was shown to improve memory performance and offered hope that photic stimulation could be used to enhance memory in conditions such as Alzheimer's Disease (Williams, 2001).

Beta frequencies (≥14Hz), on the other hand, are associated with brain activation and reflect the activity of many cortical neuronal pools. They are desynchronous rhythms (Neuper & Pfurtscheller, 2001; Niedermeyer & Lopes Da Silva, 1993; Pfurtscheller, 1992; Steriade et al., 1990) and are present during active task engagement, attentional processes (Basar-Eroglu, Stuber, Schurmann, Stadler, & Basar, 1996; Micheloyannis et al., 2002; Ray & Cole, 1985; Wrobel, 2000) and physiological arousal (Bonnet & Arand, 2001; Lim et al., 1996). They are positively correlated with anxiety (Heller, Nitschke, Etienne, & Miller, 1997; Pizzagalli et al., 2002), and inversely related to sleep quality (Nofzinger et al., 2000). Relaxation has been shown to attenuate frontal beta rhythms (Jacobs, Benson, & Friedman, 1996).

### 1.3 What is brainwave entrainment?

Brainwave entrainment or photic driving refers to "synchronisation of the EEG rhythm with the frequency identical, or harmonically related to the frequency of photic stimulation" (Wada, 1994, p. 247). Using rhythmic repetitive stimuli to entrain the brain and induce altered states of consciousness is not new. For thousands of years humankind has sought ways by which to induce relaxed states or achieve altered states of awareness in attempts to either escape from the realities of everyday life, or to access the deep recesses of the mind (Lex, 1979). Drumming, chanting, rhythmic dancing, listening to music, or staring into a campfire all have mesmerizing effects, and can be used to either arouse the nervous system or induce deep states of relaxation. What is common amongst these types of sensory stimulation is the effect they have on the brain. Rhythmic, pulsing, repetitive stimuli have the ability to entrain the brain, by enticing it to respond in kind through the production of steady state evoked potentials which are recruited and spread to distal

cortical areas, causing the cortex to behave in a synchronous manner. Thus the brain can be lulled into a state of disengagement and eventually, drowsiness. Rhythmic repetitive stimuli are the key elements of relaxation therapy as they serve to distract the mind from its incessant internal dialogue and bring it to a point of focused and relaxed awareness (Benson, 1975). Relaxation is a process of disengagement, which is evident in brainwave activity when recording the human EEG, and is quickly evoked by brainwave entrainment techniques (Glicksohn, 1986; Morse & Chow, 1993; Ossebaard, 2000; von Gizycki et al., 1998).

## 1.4 History of brainwave entrainment and photic stimulation

Hans Berger (1873-1941) was the first to describe in detail the EEG in humans. In his first publication "On the Electroencephalogram of Man" (1929), Berger described two main rhythms; a rhythm which oscillated at 10-11 hertz (Hz) which was present during rest and was later called the alpha rhythm, and a smaller faster rhythm oscillating at 20-30Hz, which he later called beta, and which was present during mental exercise and concentration, thus showing the presence of desynchronous brainwave activity during active task engagement (Berger, 1976 reprint of 1934).

Adrian and Matthews (1934) replicated Berger's work and concluded that the 'Berger rhythm', or alpha rhythm as we know it, originated in the occipital cortex and was present with the eyes closed, or with the eyes open, looking at a uniform field. In other words, occipital alpha was an indication that the brain was at 'rest', with minimal cognitive processing occurring. In contrast, higher beta rhythms were associated with stimulation of the visual cortex and denoted cortical work. In their seminal work, Adrian and Matthews were the first to show that light flicker could

produce frequency following effects in the occipital cortex. They demonstrated occipital photic driving responses to light flicker predominately in the 10Hz frequency band, but also at frequencies as high as 25Hz, and also described harmonic and sub-harmonic responses in the cortex.

The 'frequency following effect' became a well known phenomenon as subsequent researchers replicated and refined Adrian and Matthews work (Mundy-Castle, 1953; Toman, 1941; Ulett, 1953; Ulett, Gleser, Winokur, & Lawler, 1953; Ulett & Johnson, 1958; Walter & Walter, 1949). In 1941, Toman established that photic driving responses were not confined to occipital visual areas, but in some subjects were present at the vertex. While he demonstrated photic driving to light flicker from 8 to 25Hz, he found that the best responses were found in frequencies at or near the dominant alpha frequency. Toman also noted that photic driving responses had a similar morphology to auditory driving responses which had been described by Davis (1939). Davis showed that auditory driving responses were present not only in primary auditory cortex, but were capable of recruitment across the cortex.

Grey Walter (1949) was interested in the clinical applications of photic stimulation including its ability to detect abnormal brain functioning, such as epilepsy, and became a founding father of clinical neurophysiology. He was one of the first to correlate mood states with various EEG rhythms and frequencies of photically evoked brainwave patterns, and to explore the associated subjective mental imagery that often accompanies visual stimulation. He found that dysphoria correlated with parieto-temporal theta, a slow, high amplitude wave which signals cortical deactivation. In one subject he was able to induce unpleasant feelings using 11-16Hz

photic stimulation which increased theta activity via the effects of sub-harmonics. He also elucidated, in the human EEG, the effects of harmonics which are present not only in the area of cortex being stimulated, but are also recruited to other cortical regions where they give rise to sensations associated with these areas (Walter, 1953; Walter & Walter, 1949). Subsequent work by Mundy-Castle (1953) found clear evidence of recruitment of photic driving responses across the cortex and into frontal areas not only at fundamental frequencies of stimulation, but also at harmonic and sub-harmonic frequencies of the first, second and sometimes third order.

The clinical usefulness of photic driving was further elucidated by Ulett and colleagues (1953, 1958), who discovered that photic driving responses were related to anxiety levels. They found that high anxiety subjects showed greater harmonic responses in their EEG and reported feeling more uncomfortable and dysphoric during photic stimulation than their low anxiety counterparts. Shagass (1955) attempted to use photic stimulation responsivity to differentiate between anxiety and depression. He found that females showed higher photic stimulation responses than males and that anxious female subjects had greater EEG responses to 15Hz light flicker than did non-anxious depressed female subjects. He also found that photic stimulation response varied with mood states within individuals and that there was a tendency towards higher photic driving responses during anxious states, and decreased responses during relaxed states.

A common finding in the early work of photic driving and EEG was the large amount of variation in photic driving responses, across both clinical samples (Ulett, 1953; Ulett et al., 1953; Ulett & Johnson, 1958; Walter & Walter, 1949), and

between individuals (Mundy-Castle, 1953; Shagass, 1955; Toman, 1941; Walter & Walter, 1949). Some researchers found that resting alpha EEG predicted the photic stimulation response, while others did not. Ulett (1958) proposed that there was a limit to the amount of cortical reactivity possible, and that subjects with high resting alpha states would show reduced photic driving responses in comparison to subjects with low resting alpha amplitude, who had more physiological leeway. While early attempts were made to use EEG photic driving to differentiate clinical states, results were not consistent and clear diagnostic patterns did not emerge.

Another common finding among studies was that photic stimulation elicited a wide variety of subjective experiences, such as vivid visual imagery (Freedman & Marks, 1965), a disrupted sense of time, kinaesthetic sensations of swaying, rolling, or spinning, and also effects on mood states, with some subjects reporting positive changes in mood while others reported unpleasant mood changes (Mundy-Castle, 1953; Ulett et al., 1953; Walter, 1953; Walter & Walter, 1949).

In summary, early research revealed that intermittent photic stimulation could be used to 'drive' the cortex at the frequency of stimulus presentation. In the EEGs of a significant proportion of subjects studied, photic driving and harmonic responses were commonly seen, predominantly in occipital sites, and recruitment of the response across the cortex was seen in many individuals. Early research established that it was possible to use photic driving to induce a wide range of illusory sensory perceptions, vivid images, relaxation responses, and often effects on mood states. While the size of photic stimulation responses varied widely, early research showed

that photic stimulation could be used to increase EEG amplitude within particular bandwidths.

Contemporary work in the area of brainwave entrainment and EEG has replicated and vastly expanded early research and continues to explore the therapeutic utility of photic and auditory driving, its ability to influence EEG patterns, produce relaxation responses, and shift mood states (Salansky, Fedotchev, & Bonndar, 1998). Recent research has substantiated earlier claims that cortically evoked auditory or visual potentials elicited in primary sensory cortex can be used to enhance EEG power in particular bandwidths (Basar & Schurmann, 1994; Brandt, 1997; Herrmann, 2001; Schürmann, Baar-Eroglu, & Basar, 1997), which are then recruited to remote cortical areas (Dinse et al., 1997; Glicksohn, 1986; Neher, 1961; Pantev et al., 1991), increase cortical blood flow and metabolism (Diehl, Stodieck, Diehl, & Ringelstein, 1996; Sappey-Marinier et al., 1992), and also impact on mood states (Anderson, Legg, & Ridout, 1997; Brauchli, Michel, & Zeier, 1995; Lane, Kasian, Owens, & Marsh, 1998; Ossebaard, 2000; Shealy et al., 1990; Ulett, 1953; von Gizycki et al., 1998).

Today, photic driving responsivity is used for a variety of diagnostic purposes. Photic stimulation at varying frequencies is used routinely in clinical neurology to induce paroxysmal EEG activity during EEG assessment for suspected epilepsy (Diehl et al., 1996; Takahashi, 1993). Abnormal photic driving responses have been found in Alzheimer's Disease (Kikuchi et al., 2002), migraine sufferers (Chorlton & Kane, 2000; De Tommaso, Tota, Crenco, & Puca, 1996), and schizophrenics (Jin, Castellanos, Solis, & Potkin, 2000; Jin et al., 1990; Jin, Potkin, Sandman, & Bunney,

1997; Wada et al., 1995). Enhanced photic driving responses have been demonstrated in depression (Jin et al., 1997), with reduced photic driving, closer to normal levels, observed on recovery (Pockberger, Petsche, Rappelsberger, Zidek, & Zapotoczky, 1985). Photic driving responses are dependent on brain maturity and brain function and therefore change across the lifespan, with larger responses in children and the elderly in comparison to adults in their middle years (Kikuchi, Wada, Koshino, Nanbu, & Hashimoto, 2000; Niedermeyer, 1993; Takahashi, 1993). Yet, across shorter time spans, photic driving responses are relatively stable and show high test re-test reliability (Fedotchev, Bondar, & Konovalov, 1990).

It is still debatable today what baseline EEG parameters will predict photic driving reactivity. Recent research has revealed that those with higher resting alpha levels are more responsive to photic driving frequencies which resemble their own dominant resting alpha, while photic driving responses in individuals with low resting alpha frequencies tend to be smaller, and are less dependent on stimulation frequencies being in the vicinity of the peak alpha frequency (Pigeau & Frame, 1992; Rosenfeld, Reinhart, & Srivastava, 1997). In contrast, Basar and colleagues (1997) found that higher power in the 10Hz frequency band actually attenuated photic stimulation responses by 30%. Nevertheless, photic driving responses, of varying degrees, are seen in the majority of individuals, with recruitment of the response visible across the cortex (Lazarev, Simpson, Schubsky, & deAzevedo, 2001; Silberstein, 1995b).

## 1.5 Photic stimulation, relaxation and mood

The phenomenon of brainwave entrainment using photic or auditory stimuli is firmly established. However, the use of brainwave entrainment devices, such as mind-machines or brainwave synchronisers for therapeutic purposes is yet to be fully researched. To date, brainwave entrainment, often at varying frequencies of stimulation for specific clinical purposes, has been used successfully in the treatment of migraine (Anderson, 1989; De Tommaso et al., 1996), attention and behavioural disorders (Joyce & Siever, 2000; Patrick, 1996), sleep disorders (Parks, Apathy, Woodger, & Allen, 1998), mood disorders (Anderson et al., 1997; Brauchli et al., 1995; Kumano et al., 1996; Noton, 1997), anxiety reduction (Morse, 1994a; Ossebaard, 2000), enhancement of academic performance (Carter & Russell, 1993; Russell, 1997) and as tool for hypnotic induction (Kroger & Schneider, 1959). Thus there is increasing evidence that auditory and photic stimulation can be used across a wide range of conditions and that it is well tolerated by most people. These studies used a variety of mind-machines and frequencies of stimulation, but most cannot directly attribute their positive results to changes in cortical activity caused by entrainment responses, because often, measures of EEG were omitted. However, most participants reported auditory-photic stimulation to be a positive experience that produced relaxation effects.

Morse (1993, 1994) showed that brainwave synchronisers had powerful anxiolytic properties that could be used to reduce anxiety during stressful endodontic procedures. In dental patients he compared photic stimulation at 10Hz, with photic stimulation (10Hz) plus relaxing music, and a control group who received only verbal instructions. Those receiving photic stimulation with relaxing music or photic

stimulation alone, demonstrated greater relaxation responses, with decreases in heart rate, increases in skin resistance, and greater subjective reports of relaxation, than the control group (Morse, 1993; Morse & Chow, 1993).

Further exploration by Morse (1994b) found that photic stimulation at 10Hz did indeed enhance cortical alpha rhythms and produced more synchronous alpha between frontal and occipital regions with a concomitant increase in skin resistance and a decrease in heart rate, in comparison to no stimulation (Morse, 1994b). This supported Morse's earlier work (1993) and strongly suggested that the two photic stimulation groups in the previous study exhibited cortical entrainment within the alpha band alongside the demonstrated relaxation responses. While Morse's research is to be commended because he included a control group, it is difficult to determine the full extent to which placebo effects have contributed to the results. If he had included a sham condition, to control for the effect of using sophisticated equipment, which in itself may have contributed to the results, his findings would be more convincing.

In another naturalistic study, Ossebaard (2000) also found that photic stimulation had immediate anxiolytic properties, but did not appreciably impact long term on occupational burnout or anxiety over the course of the study. Forty-two employees from an addiction centre were allocated to two photic stimulation groups and exposed to either alpha (~10Hz) or beta (~25hz) stimulation and compared to a control group who received no therapy. Participants received between 7-16 sessions of photic stimulation over eight weeks, for up to 40 minutes at a time. Contrary to expectation, those receiving beta stimulation reported improvement in 'emotional

exhaustion' while those receiving alpha frequencies did not. On reflection, this is not inconsistent especially in a sample suffering from chronic anxiety and burnout. As described by Selye, after long term exposure to threat or environmental stressors, which clearly occurs in burnout, the body eventually gives up its hyper-aroused state and reaches a state of equilibrium: cortisol levels may decrease, and physiological reactivity diminishes (Chrousos & Gold, 1992; Selye, 1976, 1998). Rather than being over-aroused, individuals who are 'burnt out' are possibly beyond over-arousal, and in a state of non-reactivity. Thus beta stimulation may in fact act to 'energise' the system with a concomitant lift in mood state. As no attempt was made to measure EEG parameters in this study it was not possible to ascertain if the frequencies used in the study changed cortical functioning or merely operated as a relaxation tool by supplying 'repetitive' distracting stimuli, which induced relaxation responses.

In other research, photic stimulation was found to be more effective than autogenic relaxation in decreasing arousal levels and inducing relaxation responses. Using a repeated measures design, Dieter and Weinstein (1995) compared photic stimulation at theta frequencies with autogenic relaxation delivered via a tape, in a small sample of six people. Evidence of photic driving was found with increased amplitude in theta and delta EEG. In addition, they observed EEG characteristics, such as sleep spindles and K-complexes, which are normally only seen during sleep. According to Dieter and Weinstein, subjects remained awake throughout the thirty minutes of photic stimulation suggesting that the unique effects experienced during photic stimulation were due to the stimulation itself, and not because participants dozed off during the therapy. Participants found the experience of photic stimulation to be

pleasant, and reported marked changes in mood and accompanying visual imagery, which were not present during autogenic relaxation.

These findings are consistent with other studies which also found that photic stimulation reduced arousal, increased drowsiness, and produced hypnagogic type images similar to those experienced during the early stages of sleep (von Gizycki et al., 1997; von Gizycki et al., 1998). Von Gizycki showed that photic stimulation impacted on mood, but not necessarily towards a positive valence, but simply because it increased sleepiness and reduced arousal. Similarly to Dieter and Weinstein, von Gizycki concluded that photic stimulation produced altered states of consciousness that resembled the early stages of sleep, thereby making it a potentially useful tool in the treatment of sleep onset insomnia.

Brauchli and colleagues (Brauchli, 1993; Brauchli et al., 1995) offer further support for the mood enhancing and stress reducing qualities of photic stimulation. In an initial study Brauchli (1993) compared audiovisual stimulation with a simple relaxation method, such as listening to taped 'sounds of nature'. In this study, both relaxation and audiovisual stimulation effectively reduced physiological arousal with decreases in muscle tension, heart rate, skin conductivity, and a reduction in salivary cortisol. Greater mood effects were found for audiovisual stimulation with participants reporting feeling 'warmer and calmer', in comparison to simple relaxation, but no differences were found between the two methods in their ability to induce relaxation responses. In this study, it is not possible to attribute the anxiolytic and mood enhancing qualities of audiovisual stimulation to entrainment, as EEG was not recorded, and while immediate relaxation responses were found for both

techniques, the long term effects of audiovisual stimulation and relaxation on mood and arousal were also not assessed.

In subsequent research, Brauchli, Michel, and Zeier (1995) found that audiovisual stimulation of varying frequencies, patterns, and intensities impacted differentially on subjective mood. They exposed 20 male subjects to three different audiovisual programs, *i*) a 'low' variation program, with a constant and stable audiovisual frequency presented to both eyes simultaneously, *ii*) a 'high' variation program comprising a high degree of variability in audiovisual frequencies, temporal presentations of stimuli, pattern of light flicker, stimulus intensity, and shifts between right and left visual presentation, *iii*) a 'high-low' program, which shifted between high and low variation programs. Participants preferred the combination of high and low variation, because it was variable and maintained interest, but was not over stimulating. Subjects reported feeling more 'warm', 'comfortable', 'peaceful', and 'calmer', after the high-low program, but in contrast, felt 'bored' after the low-no variation program, and 'aroused' after the high intensity and very changeable, high variation program.

It is difficult to assess direct entrainment effects in this study, as it assessed the impact of highly variable patterns of audiovisual stimuli on the EEG. Alpha attenuation was observed across all audiovisual programs indicating cortical activation. This was possibly related to a combination of auditory-photic driving responses and cognitive processing of the novel sensory stimuli being presented. Nevertheless, auditory-photic stimulation that was novel and not overly arousing, induced positive mood changes after a short program of only seven minutes.

Further evidence for the use of photic stimulation to enhance mood was found when positive mood changes were noted in a sample of seventeen women using photic stimulation at 30Hz for the treatment of pre-menstrual disorder (Anderson et al., 1997). Anderson et al. reported a 76% decrease in pre-menstrual symptoms, with significant reductions in physical and cognitive symptoms, depression, anxiety, fatigue, and irritability, after 3months of regular photic stimulation for at least fifteen minutes a day. This study, however, was limited in a number of ways. Firstly, because there were no EEG measures, the presence of entrainment effects could not be assessed. Secondly, because of the omission of a control group, it was not possible to ascertain the extent to which placebo effects contributed to the effects observed. Nevertheless, Anderson did observe significant decreases in symptoms, including reductions in anxiety and depression, possibly due to a combination of cortical entrainment effects, placebo effects, and relaxation responses induced by photic stimulation.

In contrast, Walach (1998) found that audiovisual stimulation, and cranial electric stimulation, were no better than placebo at enhancing well-being and eliciting relaxation responses. Employing a repeated measures design, thirty six volunteers were exposed to audiovisual stimulation (at 7.83Hz), cranial electrical stimulation (at 7.83Hz) and sham stimulation (electric stimulation device without stimulating) for twenty five minutes, over a number of days. All conditions elicited relaxation responses, including the baseline rest condition, with decreases in skin conductance observed after each session, but no differences were observed between groups. Audiovisual stimulation, however, produced more mystical experiences with subjects

reporting a diffusion of boundaries between the self and the environment, and reduced 'emotional tension', in comparison to those exposed to cranial electrical stimulation or sham stimulation.

Placebo effects in this study were significant with subjects in the sham condition reporting relaxation responses similar to active experimental conditions. Other research has shown that the use of sophisticated equipment can elicit strong placebo effects which can match active treatments (Ho, Hashish, Salmon, Freeman, & Harvey, 1988). A shortcoming in Walach's study, however, was the omission of EEG evidence to determine if the entrainment devices used were actually 'entraining' the brain and producing enhancements in cortical activity at desired frequencies.

In summary, both early and contemporary research have shown that the cortex can be entrained using light or sound pulses of varying frequencies. A major assumption of brainwave entrainment therapy is that by artificially driving the brain into particular rhythms, particular mood states, which are usually associated with these rhythms, will be generated. For example, if the brain is coaxed into increased synchrony within the alpha bandwidth, then a subjective state of relaxed awareness should follow. It is well established that audiovisual stimulation produces vivid visual imagery in many people (Freedman & Marks, 1965; Glicksohn, 1986; Richardson & McAndrew, 1990; Stwertka, 1993), and that theta or alpha rhythms often elicit physiological relaxation with accompanying feelings of drowsiness, 'letting go', and increased calm (Dieter & Weinstein, 1995; Joyce & Siever, 2000; Kumano et al., 1996; Morse, 1993, 1994a; Ossebaard, 2000; Parks et al., 1998; Rosenfeld, 1997;

Rosenfeld et al., 1997; Shealy et al., 1990; von Gizycki et al., 1997; von Gizycki et al., 1998).

The therapeutic utility of photic stimulation, however, remains equivocal. Some studies, for example (Dieter & Weinstein, 1995), have shown audiovisual stimulation to be superior to traditional relaxation methods in its ability to produce relaxation responses and induce positive mood states, while others, such as Walach (1998), have not. Other studies have used only very short exposure times to entrainment stimuli and shown that photic driving elicits immediate mood and relaxation effects (Brauchli et al., 1995; von Gizycki et al., 1997; von Gizycki et al., 1998), while other studies have employed repeated measures designs with longer exposure times and found photic stimulation to be no better than placebo (Walach, 1998). Brainwave synchronisers have been shown to have immediate anxiolytic effects (Morse, 1993, 1994a; Ossebaard, 2000), but there is limited support for their ability to produce long-term, enduring therapeutic changes in the psychologically distressed (Ossebaard, 2000).

In the therapeutic arena, it is widely accepted that relaxation is a skill that requires time, practice, and effort to achieve mastery. Practice facilitates 're-training' of the autonomic nervous system by repeatedly activating parasympathetic responses, reducing sympathetic activation, and re-establishing homeostatic balance (Argras, Taylor, Kraemer, Allen, & Schneider, 1980; Benson, 1975, 1985; Lehrer, 1997; Lehrer et al., 2000). In contrast to traditional relaxation therapies, photic stimulation requires no effort to attain mastery. Rather, it effortlessly elicits a relaxation response using a centrally mediated mechanism of brainwave entrainment, as

opposed to relaxation techniques such as progressive muscle relaxation, which use peripherally mediated mechanisms. Couple this with important elements of relaxation, such as distracting rhythmic stimuli, isolation from external environmental cues, and reduced muscle tone which accompanies the relaxation response, and brainwave entrainment techniques have the potential to be powerful clinical tools.

## 1.6 Summary

Mind machines which use light and sound stimuli for brainwave entrainment are commercially available, portable, relatively inexpensive, and offer a quick and easy means of providing treatment for stress and stress related disorders in a wide range of contexts. In addition, in a busy modern world that is fascinated by technology they are attractive and effective coping tools that can assist with the management of increasing daily stressors. The popularity of these devices, however, exceeds the research to support their effectiveness. While much of the current research using brainwave synchronizers reports them to be effective across a wide range of clinical applications, few studies have used control groups in order to gauge the extent to which placebo effects contribute to reported results. When control groups have been included, often expectancy effects are left un-addressed. The inclusion of a sham condition would effectively address this issue. In addition, other procedures, which improve experimental rigor, are often missing from clinical research, such as random allocation to experimental groups, and double blind procedures to control for experimenter bias.

In order to validate the use photic stimulation for therapeutic purposes, it is firstly necessary to address some of these shortcomings. In the next chapter, two important questions are asked; first, 'Are brainwave synchronizers as effective as traditional relaxation techniques in their ability to induce relaxation responses?' and second, 'if they do produce relaxation effects, are these effects dependent on demonstrable cortical driving responses, or does brainwave entrainment merely act as a relaxation aid because it provides a distracting repetitive rhythmic stimulus, and assists to minimise sensory input from environmental sources?'

# CHAPTER 2

## Study 1: 'Effects of audiovisual stimulation on mood, arousal, and EEG'.

### 2.1 Introduction

Previous research has found that photic stimulation can elicit relaxation responses and induce different mood states (Brauchli et al., 1995; Dieter & Weinstein, 1995; Glicksohn, 1986; Mundy-Castle, 1953; Noton, 1997; Richardson & McAndrew, 1990; von Gizycki et al., 1998). Through the process of entrainment, photic stimulation acts to captivate cortical EEG, and pull it into synchrony with the presenting stimulus. As a result, EEG power is increased, not only at the frequency of stimulation, but also within distal bands of EEG harmonically related to the original stimulus, with concomitant changes in mood and physiological arousal.

As discussed in Chapter 1, many studies attest to the anxiolytic effects of photic stimulation, and have shown that it quickly induces relaxation responses, and increases brainwave activity in alpha and theta bandwidths, which are EEG rhythms associated with relaxation and cortical deactivation (Brauchli, 1993; Dieter & Weinstein, 1995; Kumano et al., 1996; Morse, 1993, 1994b). Morse investigated the anxiety reducing abilities of photic stimulation in patients undergoing root canal therapy. He compared 10Hz photic stimulation, photic stimulation combined with relaxing music, and a control group, who received no additional intervention. Those who chose to use the brainwave synchroniser, with or without relaxing music, demonstrated significantly greater relaxation responses, even during a potentially stressful procedure. Dieter and Weinstein (1995) found photic stimulation to be superior to autogenic relaxation, as it produced deeper relaxation, increased theta and delta EEG activity, plus induced vivid visual imagery similar to that found in the

hypnagogic state. This study was consistent with other research which found that photic stimulation increased drowsiness and produced altered states of consciousness, with the potential to be an effective tool for treating insomnia (Glicksohn, 1986; von Gizycki et al., 1997; von Gizycki et al., 1998).

Other studies have assessed the therapeutic effectiveness of mind machines on arousal and psychological distress. Anderson, Legg and Ridout (1997) demonstrated that daily, long term use (2-4 months) of photic stimulation dramatically reduced premenstrual symptoms in a group of women suffering from premenstrual dysphoric disorder. Conversely, Ossebaard (2000) found that audiovisual stimulation, presented twice a week, for up to eight weeks, reliably produced immediate relaxation effects during stimulation, but did not have an enduring impact on psychological distress in a group suffering from occupational burnout.

Much of this research, however, used either single trial paradigms, or short exposure times to the entraining stimulus, or both. Research which has attempted to assess the therapeutic effects of photic stimulation of longer duration (15-40 mins) with repeated exposures (7-60 sessions) were either methodologically flawed because of omission of control groups (Anderson et al., 1997; Noton, 1997), or found photic stimulation gave immediate benefit, but failed to produce long-term therapeutic change over time (Ossebaard, 2000). There is a paucity of research investigating the effects of repeated exposure to an entrainment stimulus of longer duration on psychological and physiological variables, while controlling for placebo and relaxation effects, in order to fully assess the therapeutic utility of AVS. To date, there is little evidence to show that photic stimulation, like relaxation therapy,

produces carry-over or training effects which create permanent reductions in arousal mechanisms, thereby re-establishing physiological homeostasis, and also substantiating its effectiveness in the treatment of arousal disorders.

To address some of the shortcomings of previous studies the current study will include the following, *i*) randomised allocation of participants to experimental trials, *ii*) control of relaxation and placebo effects with the inclusion of control and relaxation conditions, and *iii*) use of a repeated measures design over many sessions in order to fully assess the clinical utility of brainwave entrainment techniques.

This initial study adds to previous research by exposing participants to extended periods of various AVS frequencies, over a number of trials, while measuring key physiological variables known to reliably reflect changes in arousal; such as heart rate (Lucini et al., 1997; Sakakibara, Takeuchi, & Hayano, 1994; Scher, Furedy, & Heslegrave, 1985; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002), skin conductance (Heller, Nitschke, & Lindsay, 1997; Lim et al., 1996; Montgomery, 1998), EEG; and also mood, in an attempt to see if prolonged and regular exposure to photic stimulation will have enduring effects on mood and arousal. Consequently, if photic stimulation can be shown to have beneficial and enduring effects on mood, with concomitant reductions in physiological arousal, this will pave the way for the use of entrainment technologies as clinical tools in the treatment of arousal and mood disorders.

## 2.2 Aims of Study 1

As discussed in Chapter 1, photic driving is a well established phenomenon (Mundy-Castle, 1953; Toman, 1941), which can be used to reduce arousal and generate different mood states by 'driving' the brain into particular rhythms (Dieter & Weinstein, 1995; Richardson & McAndrew, 1990; von Gizycki et al., 1998). The current study employed a single blind, controlled, repeated measures design to investigate the effects of three audiovisual frequencies, 5Hz (theta), 13Hz (alpha), and 22Hz (beta), on mood and physiological arousal, in comparison to relaxation.

There were two main aims of this initial study. First, to determine if photic stimulation is superior to a simple relaxation technique, such as autogenic relaxation, in its ability to produce relaxation responses with enduring effects over time. Second, to ascertain which particular photic stimulation frequencies are more conducive to the production of relaxation responses and positive mood states.

## 2.3 Hypotheses

**2.3.1 Heart rate, skin conductance, & subjective relaxation:** Reductions in physiological arousal were anticipated for all participants, as they were required to sit quietly in a darkened room for up to forty minutes during each recording session. As discussed in Chapter 1, EEG frequencies in the theta and alpha ranges signal cortical deactivation (Niedermeyer, 1993; Steriade et al., 1990), and are present during relaxed states (Aftanas, Varlamov, Pavlov, Makhnev, & Reva, 2002; Marshall & Bentler, 1976; West, 1980). Beta frequencies, on the other hand, indicate cortical arousal and are often associated with physiological arousal and anxiety (Bonnet & Arand, 2001; Nofzinger et al., 2000).

In the current study, therefore, it was anticipated, that brainwave entrainment at alpha and theta frequencies would induce greater relaxation responses than entrainment within the beta bandwidth. Given that relaxation is associated with alpha and theta states, it was predicted that the theta, alpha, and relaxation groups would experience greater reductions in arousal, as evidenced by reduced heart rate and skin conductance during the active experimental phase, than the beta or control groups. Congruent with this hypothesis, it was anticipated that the theta, alpha and relaxation groups would report higher levels of subjective relaxation than the beta or control groups, across experimental sessions.

**2.3.2 Mood, automatic thinking, and psychological status:** In alignment with previous research which found that theta and alpha rhythms in particular, often elicit relaxation responses and positive, pleasant feeling states (Anderson et al., 1997; Brauchli, 1993; Brauchli et al., 1995; Brown, 1970, 1971; Glicksohn, 1986; Kumano et al., 1996; Morse, 1993; Noton, 1997; Nowlis & Kamiya, 1970; Shealy et al., 1990; von Gizycki et al., 1997; von Gizycki et al., 1998) it was predicted there would be greater mood enhancements for the alpha, theta and relaxation groups than for the beta and control conditions.

In addition, it was anticipated that as relaxation responses were elicited and mood shifted towards a more positive valence, that automatic thinking would also shift towards the positive pole. Negative automatic thoughts often accompany dysphoria and are the object of therapy in cognitive behavioural models (Beck, 1995). As mood shifts towards a more positive valence, concomitant shifts in thinking are also

observed (Abela & D'Alessandro, 2002; Ellis & Harper, 1961; Kendall, 1992; Kendall, Howard, & Hays, 1989; Smith, Haynes, Lazarus, & Pope, 1993). If changes in automatic thinking can be demonstrated alongside shifts in mood towards the positive valence in the current study, using a non-clinical sample, this would give further credence for the utility of brainwave entrainment techniques as therapeutic tools. Given the superiority of theta and alpha rhythms to bring forth relaxation responses, and their association with positive mood states, it was anticipated that the theta, alpha, and relaxation groups would experience greater reductions in negative affect and negative thinking, and larger increases in positive affect and positive thinking, than the beta and control groups during experimental trials. Concomitantly, it was predicted that depression, anxiety, and symptom severity scores, as measured by the Symptom Checklist 90-R (Derogatis, 1977), would decrease more for the theta, alpha, and relaxation groups, than for the beta and control conditions.

**2.3.3 Electroencephalography (EEG)**: The response of the occipital cortex to light flicker has been well documented with photic driving observed in response to frequencies between 5 to 90Hz (Fedotchev et al., 1990; Herrmann, 2001; Lazarev et al., 2001; Mundy-Castle, 1953; Silberstein, 1995b; Takahashi, 1993; Toman, 1941). Optimal driving responses, however, are usually seen at frequencies nearest to the clients' own dominant alpha frequency which lies within the 8-12Hz range (Pockberger et al., 1985; Toman, 1941). Therefore, it was anticipated that evidence of frequency following would be seen in the posterior cortex at entrainment frequencies (5Hz, 13Hz or 22Hz), with observable increases from baseline in EEG magnitude at the stimulus frequency.

Finally, the study aimed to assess if repeated and regular exposure to photic stimulation would produce carryover or 'training effects' persisting beyond the intervention sessions, with sustained positive changes in mood status and decreases in heart rate and skin conductance. It was anticipated that entrainment responses, that is increased EEG amplitude at frequencies of stimulation, would be contingent on the stimulus itself. However, if evidence of increased magnitude in occipital EEG at entrainment frequencies is found at follow-up, in the absence of photic stimulation, this would give further validation for the use of photic stimulation as a clinical tool, showing enduring EEG changes which persist beyond the intervention phase.

## 2.4 Method

**2.4.1 Participants:** The sample comprised thirty adult participants, 10 males and 20 females, drawn from the pool of first year psychology students at the Australian National University, and from the wider community. Many respondents received course credit points for their participation. Participants from the wider community responded to advertisements placed around the university campus, or placed in a number of local health clinics. The age range of participants was from 18 to 59 years, with 70% of participants aged 32yrs or younger.

In order to minimise risk to participants, remove the effects of medication on EEG recordings, and, as much as possible, to obtain a sample free from psychopathology, participants were excluded from the study if they had a history of epilepsy or if they were currently taking antidepressant, sedative or neuroleptic medication. Nevertheless, 17% (n=6) of participants had Symptom Checklist 90-R (SCL-90-R)

depression T-scores greater than 60, and 27% (n=9) had SCL-90-R anxiety T-scores greater than 60. In addition, at baseline 10% (n=4) of participants had a global severity index T-scores ≥ 63, thus identifying them as 'positive risks' for pathology (Derogatis, 1977).

## 2.5 Design

A 5x14 mixed, single blind, controlled design was employed. The thirty participants were randomly allocated to one of 5 groups; a control group who received no active stimuli, a relaxation group who listened to a 20 minute autogenic relaxation tape, or one of three audiovisual stimulation (AVS) groups: theta, alpha or beta, who received stimulation at the following frequencies 5Hz, 13Hz and 22Hz respectively. Initial baseline measures were obtained, followed by 12 intervention sessions, and finally, a follow-up session two weeks after completion of the experimental phase.

Measures of electroencephalography (EEG), skin conductance, heart rate, subjective relaxation, daily hassles and mood were taken at all sessions. Additional measures of psychological status (depression, anxiety, and symptom severity), and positive and negative automatic thoughts were obtained at baseline and follow-up sessions.

## 2.6 Measures

Demographic information, measures of mood, automatic thoughts, daily hassles, subjective relaxation, and health behaviours and were presented together in the 'Thoughts Emotions and Health Questionnaire' (see Appendix A). On subsequent assessments a shorter version of this questionnaire, which omitted previously

obtained demographic details, was used. Specifically, the questionnaire assessed the following variables:

**2.6.1 Mood**: Mood was assessed using the Positive and Negative Affect Schedule (PANAS) developed by Watson and Clark (1988) (see Appendix A, 'Thoughts, Emotions and Health Questionnaire', p. 1). This is a 20 item scale with 10 items assessing positive affect and 10 items assessing negative affect. Respondents were asked to indicate, on 5 point Likert scale (1= very slightly or not at all; 5= extremely) the extent to which they endorsed each item. At baseline and follow-up participants were instructed to indicate the extent to which each item described how they felt 'on average in the last week'. During experimental trials, sessions 2-12, participants were instructed to indicate the extent to which each item described how they felt on average since the last session.

Positive and negative affect are purported to be orthogonal constructs (Diener & Emmons, 1985; Watson, Clark, & Tellegen, 1988). Factor analysis of the schedule across a number of administrations, was generally consistent with this with a strong positive factor emerging, and a weaker dichotomous negative factor which often comprised an additional lower-order 'arousal' type factor containing the adjectives 'afraid', 'scared' or 'nervous'. This finding is consistent with Molloy (2001) and others who also found positive affect to be robust and negative affect to be comprised of an additional 'afraid' type factor (Glenn & Molloy, 2000; Killgore, 2000; Molloy, Pallant, & Aristotle, 2001).

The alpha reliability coefficients for the two scales in this current study were 0.90 for positive affect and 0.87 for negative affect (Cronbach, 1951). Correlations between negative and positive affect confirmed their independence in all but one analysis. At initial assessment a significant negative correlation was found ($r = -.57$, $p < .05$) between the two subscales. However, all subsequent correlation coefficients showed no relationship between positive and negative affect with an average correlation of $r = 0.03$ (range $r = -0.10$ to $0.16$) $p > .05$.

**2.6.2 Daily Hassles**: Daily hassles were assessed at each session because of their ability to impact on mood states. A selection of 32 central daily hassles were used from Gruen, Folkman and Lazarus' review of 'central daily hassles' (Gruen, Folkman et al. 1988) (see Appendix A, 'The Thoughts, Emotions and Health Questionnaire', p. 3). Respondents were told that "a 'hassle' is an irritant which annoys or bothers you; they can make you upset or angry, they can be minor irritants or fairly major pressures." They were then asked to indicate on a 5 point Likert scale (0 = none or not applicable, through to 4 = a great deal) "the extent to which each hassle has been or currently is problematic". Scores were then summed to obtain a 'hassle intensity' score, range 0-99, for each session.

**2.6.3 Automatic Thoughts:** As discussed above, automatic thinking was assessed in this study because of its association with mood. Positive mood states tend to be accompanied by positive automatic thinking, while negative mood states are characterised by negative automatic thinking. Positive and negative automatic thoughts were assessed at baseline and follow-up using both the Automatic Thoughts Questionnaire- Positive (ATQ-P), devised by Ingram and Wisnicki (1988), and the

original Automatic Thoughts Questionnaire (ATQ), by Hollon and Kendall (1980), which assesses negative automatic thinking. In total both scales comprised 60 items, 30 positive and 30 negative self-referential thoughts, which were presented in a counterbalanced fashion. Respondents were asked to indicate on a 5 point Likert scale (1 = not at all, 5 = all the time) the frequency that each thought had occurred over the last week (see Appendix A, 'Thoughts, Emotions and Health Questionnaire', p. 2).

Visual examination of baseline and follow-up scree plots showed two dominant factors. Factor analysis showed the presence of a strong positive thoughts factor at both baseline and follow-up. Inspection of the un-rotated principal components matrix showed all of the 30 positive baseline items loading strongly on the positive thoughts factor, with only one item, 'I won't give up', failing to load at follow-up. The factor structure for negative thoughts was less robust across the two measurements. At baseline, 'negative thoughts' was comprised of one main factor and two lower order factors. Varimax rotation did not improve item loadings. At follow-up, a more robust negative thoughts factor was observed with a main factor and two smaller lower-order factors comprising the items, 'my life's not going the way I want', 'I wish I were somewhere else', 'I've let people down', and a final factor with only one item; 'something has to change'. At follow-up only one negative thoughts item, 'I can't get started', failed to load on either scale.

Alpha reliabilities for positive thoughts were 0.97 at baseline and 0.98 at follow-up and for negative thoughts, 0.88 at baseline and 0.87 at follow-up. Little

improvement in internal consistency of the scales was achieved with the removal of low-loading items, therefore all items were retained for final analysis.

**2.6.4 Psychological status:** The Symptom checklist 90-R (SCL-90-R) (Derogatis, 1977) was used to assess the psychological status of study participants and to assess the extent to which psychopathology may be present. The SCL-90-R is a 90 item inventory which has been extensively used on both 'normal' and clinical populations. It measures nine subscales including, Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychotocism. It also includes three global symptom severity indices, the Global Symptoms severity index, the Positive Symptom Distress Index, and Positive Symptom Total. Respondents were asked to indicate on a 5 point Likert scale the extent to which they had been distressed or bothered by each of the 90 listed problems over the last 7 days.

The SCL-90-R reports good reliability and validity with alpha coefficients for internal consistency reported for the depression scale at 0.90 and for the anxiety scale, 0.88, and high test-retest reliability, 0.82 (Derogatis & Savitz, 1999). The subscales used in this study were depression, anxiety, and the global severity index and are measured as T-scores with mean of 50 and SD of 10.

**2.6.5 Lifestyle variables:** Lifestyle variables, which have been shown to impact on mood, were assessed at baseline and follow-up sessions only. Respondents were asked to indicate if they had suffered any medical condition/s which had resulted in hospitalisation during the last year and experienced any major life events during the

last three months. Information was also sought about other lifestyle factors such as alcohol consumption, smoking behaviour, and the use of both prescribed and recreational drugs. In addition, given that it is widely reported that the phase of the menstrual cycle also impacts on mood (American Psychiatric Association, 1995; Mackenzie, Wilcox, & Baron, 1986), women were asked, at all sessions, to indicate the current week (1-4) of their menstrual cycle. Week one corresponded to the onset of menses, followed by subsequent weeks 2 and 3, with week 4 indicating the final week of the cycle or encompassing the remaining time until the beginning of the next cycle (see Appendix A, 'Thoughts, Emotions & Health Questionnaire', p. 3 - 4).

Eighty seven percent of the sample reported that they had not had a medical condition in the last year resulting in hospitalisation. Forty per cent of the sample reported they had experienced a major life event in the last three months prior to commencing the study, such as university examinations, relationship break-ups, or stress related to work and finances. Most of the sample consumed alcohol less than twice per week and only 17% smoked ten or more cigarettes per day. Only seven (27%) of this predominately student population, reported using recreational drugs, with most of these claiming to use less than once per month.

**2.6.6 Subjective relaxation:** Subjective relaxation was assessed at each session. Participants were asked to indicate on a 10 point Likert scale (1 = feeling very relaxed and calm, to 10 = feeling very anxious and uptight) how relaxed and calm, or anxious and uptight, they felt 'right now'. In order to obtain a measure for use in statistical analysis that was reflective of the descriptor 'subjective relaxation', this variable was reverse scored so that high scores corresponded with high levels of

relaxation and low scores with low scores of relaxation (see Appendix A, 'Thoughts, Emotions & Health Questionnaire', p. 5).

## 2.7 Apparatus

All physiological data were recorded for twenty minutes at each session using an AMLAB data acquisition system with version 2.01B software. All data were sampled at 100Hz and saved to hard drive for subsequent analysis.

**2.7.1 Electroencephalogram (EEG):** EEG was recorded using gold-cup Grass electrodes placed with Elefix™ electrode paste. All sites were prepared with alcohol prior to electrode placement in the following sequential montage, F3-Fp1, F4-Fp2, P3-O1, P4-02, and referenced to the right ear, according to the 10-20 International System (Jasper 1958; Homan, Herman et al. 1987). A 40Hz low-pass filter was used. Electrode impedances were kept below 10Kohms (Andreassi, 1995). See 'Data Analysis' below, for a description of artifact removal and data reduction methods.

**2.7.2 Heart rate:** Heart rate (beats per minute) was recorded using standard disposable pre-gelled 3M™ silver-silver chloride (Ag/AgCl) electrodes. Electrodes were placed mid sub-clavicular on the left and right hand side of the chest wall with a left mid clavicular electrode placed over the 5-6$^{th}$ inter -costal space. The skin was prepared with alcohol prior to electrode placement.

**2.7.3 Skin conductance**: Skin conductance was measured in micromhos and recorded using a pair of Ag/AgCl 'wet' skin conductance sensors filled with .05M

sodium chloride gel and applied to the volar surfaces of the 2nd and 3rd middle phalanges of the left hand.

**2.7.4 Audiovisual stimulation (AVS):** Because of the claim, made by Mind Machine manufacturers, that coupled photic and auditory stimulation enhances entrainment effects, audiovisual stimulation (AVS) was used in the current study. A portable light and sound machine (XCELR8R II) manufactured by 'Mind-gear'™ PTY. LTD was used to deliver audiovisual stimulation of either 5Hz, 13Hz, or 22Hz. White light was delivered via a set of goggles with integrated white light emitting diodes set at 25% intensity (~ 8-10 Lux) in an attempt to obtain a level of light intensity tolerable by all participants. Auditory stimulation consisted of a pulsed single tone delivered simultaneously to both ears and was contingent with the light frequency and delivered via a set of headphones at a volume audible and comfortable for the client.

**2.7.5 Autogenic Relaxation**: Participants in the relaxation group listened to twenty minutes of taped autogenic relaxation instructions delivered via the Mind-gear headphones. Tape instructions firstly asked participants to focus on the breath and to gradually slow and deepen their breathing. The adoption of a passive attitude to incoming thoughts was encouraged. Instructions then guided listeners through the body, from the head to the toes, focusing on letting go of tension and noticing areas of relaxation and calm. Finally, towards the end of the 20 minutes focus was again brought back to the breath and awareness placed on external stimuli and the client brought to an awake state (see Appendix B for relaxation script). Participants were

seated semi-reclined in a chair, eyes closed with their legs resting on a footstool and wearing headphones and the light goggles but with no visual stimulation.

## 2.8 Procedure

Participants were seen individually and all sessions were conducted in a laboratory in the Psychology Department at the Australian National University. In order to maximise the effects of relaxation responses participants were asked to attend their 12 intervention sessions up to 3 to 4 times a week over a three to four week period. In addition, because of 'time of day' effects on the EEG, sessions were scheduled as much as possible at the same time each day (Cummings, Dane, Rhodes, Lynch, & Hughes, 2000; Sterman, 1998). While every attempt was made to adhere to this regime, not all participants could comply.

Prior to assessment, participants were informed that the study was investigating the effects of relaxation and AVS on mood and other physiological variables. To control for expectancy effects all participants were told that there had been reports about the beneficial effects of AVS for the treatment of stress and other disorders and that the current research was being conducted because there was a paucity of controlled research to validate these claims. The procedure of 'random allocation to experimental groups' was explained. Participants were told they would not know the frequency of AVS stimulation they would receive, or the intended effects of this stimulation, until the end of the study. Recording procedures were explained, participants rights discussed, and informed consent obtained. Participants completed the 'Thoughts Emotions and Health Questionnaire' prior to the placement of electrodes and recording of physiological measures. Participants were seen

individually over a period of 6-8 weeks for each subject with data collection for all subjects extending over a 9 month period (November 1997 to July, 1998).

In order to minimize arousal induced by attachment of recording equipment and the novelty of the situation, the first session included a twenty minute habituation phase. After attachment of sensors and testing of equipment, participants were instructed to sit quietly for twenty minutes with their eyes closed. Subsequent sessions included only ten minutes of habituation prior to recording as participants were more familiar with the experimental milieu and procedures (Andreassi, 1995).

Participants were seated in an armchair in a semi-reclined position with their feet supported on a footstool, in a light attenuated room. All participants wore headphones and light goggles and sat with their eyes closed throughout the twenty minute recording period. During this time the control group received no stimulus, the relaxation group received instructions via the headphones, but no photic stimulation, while the AVS groups received pulsed sound via the headphones and photic stimulation via the light goggles at either 5Hz, 13Hz, or 22Hz. The experimenter was present in the same room throughout the recording procedure, seated away from the participant and observing occipital EEG tracings for evidence of increased slow wave activity that often precedes the onset of drowsiness. All participants were cued to stay awake ten minutes into the recording session, if however, there appeared to be an increase in slow wave activity in occipital leads at any time during the recording session, further cues were given.

## 2.9 Results

**2.9.1 Data analysis**: To minimize the effects of artifact contamination the first and last minute of EEG data was ignored. Data were sampled from five equidistant points that were determined a priori on the remaining 18 minutes of EEG trace. Left pre-frontal (Fp1-F3) raw EEG tracings were visually assessed by a trained technician for removal of eye movement and other artifact. Artifact contaminated EEG was removed from all leads at the same corresponding time epochs and the remaining raw EEG was subject to a Fast Fourier Transform. Further data reduction involved averaging across three consecutive 5.5 second epochs at or close to the five predetermined sampling points, yielding a total of 80-90 seconds of artifact free EEG from each session. In order to assess the effects of the three AVS frequencies (5Hz, 13Hz, and 22Hz) on the EEG, three EEG variables encompassing these bandwidths were formed, *i*) 4-6Hz, *ii*) 12-14Hz, and *iii*) 21-23Hz. Similarly, heart rate and skin conductance data were extracted at the same 'time epochs' that the EEG data were collected.

The assumptions of linearity, normal distribution and homogeneous variance, were checked graphically by means of residual plots. The data were also screened for outliers using Mahalanobis's distance and visual inspection of box plots. Original EEG data was measured in decibels (dB). This data tended to be positively skewed, but transformation into microvolts (μV) rendered the data more 'normal'. The transformed data were used in subsequent analysis. Data analysis was done using the SPSS V.10 statistical package. Repeated measures multivariate analyses of variance (MANOVA) was used to test hypotheses. Means and standard deviations for variables used in each MANOVA are located in Appendix C.

**2.9.2 Heart rate & skin conductance:** The main aim of this study was to determine if AVS was superior to relaxation in its ability to enhance mood and induce a relaxed state. Because of the length of time participants were required to sit quietly with their eyes closed it was predicted that all groups would experience a decrease in physiological arousal with decreases in heart rate and skin conductance over the twenty minute recording time (time effect).

It was also hypothesised there would be greater reductions in heart rate and skin conductance for the theta, alpha, and relaxation groups compared to the beta and control groups across experimental sessions. To test these hypotheses separate three-way MANOVA's were computed with group (control vs. relaxation vs. theta vs. alpha vs. beta) as the between groups variable, and time (sampling time 1-5) and session (1-14) as within-groups variables for both heart rate and skin conductance. There were no differences between groups at baseline for either heart rate or skin conductance.

Analysis showed the expected decreases in physiological arousal over the twenty minute recording period with a significant main effect for time for both heart rate, $F(4,20) = 11.30$, $p < .05$, $\eta^2 = .69$, and skin conductance, $F(4,22) = 14.40$, $p < .05$, $\eta^2 = .72$. This effect was not influenced by group membership as indicated by non-significant group by time interactions for both heart rate, $F(16,92 = .93$, $p > .05$, $\eta^2 = .14$, and skin conductance, $F(16,100) = 1.70$, $p > .05$, $\eta^2 = .21$. As can be seen in Figure 2.1, contrary to predictions, mean heart rate increased over the fourteen sessions for all groups regardless of a decrease during each individual recording

period, however this moderate to large main session effect was not statistically significant, $F(13,11) = 2.10$, $p > .05$, $\eta^2 = .71$.

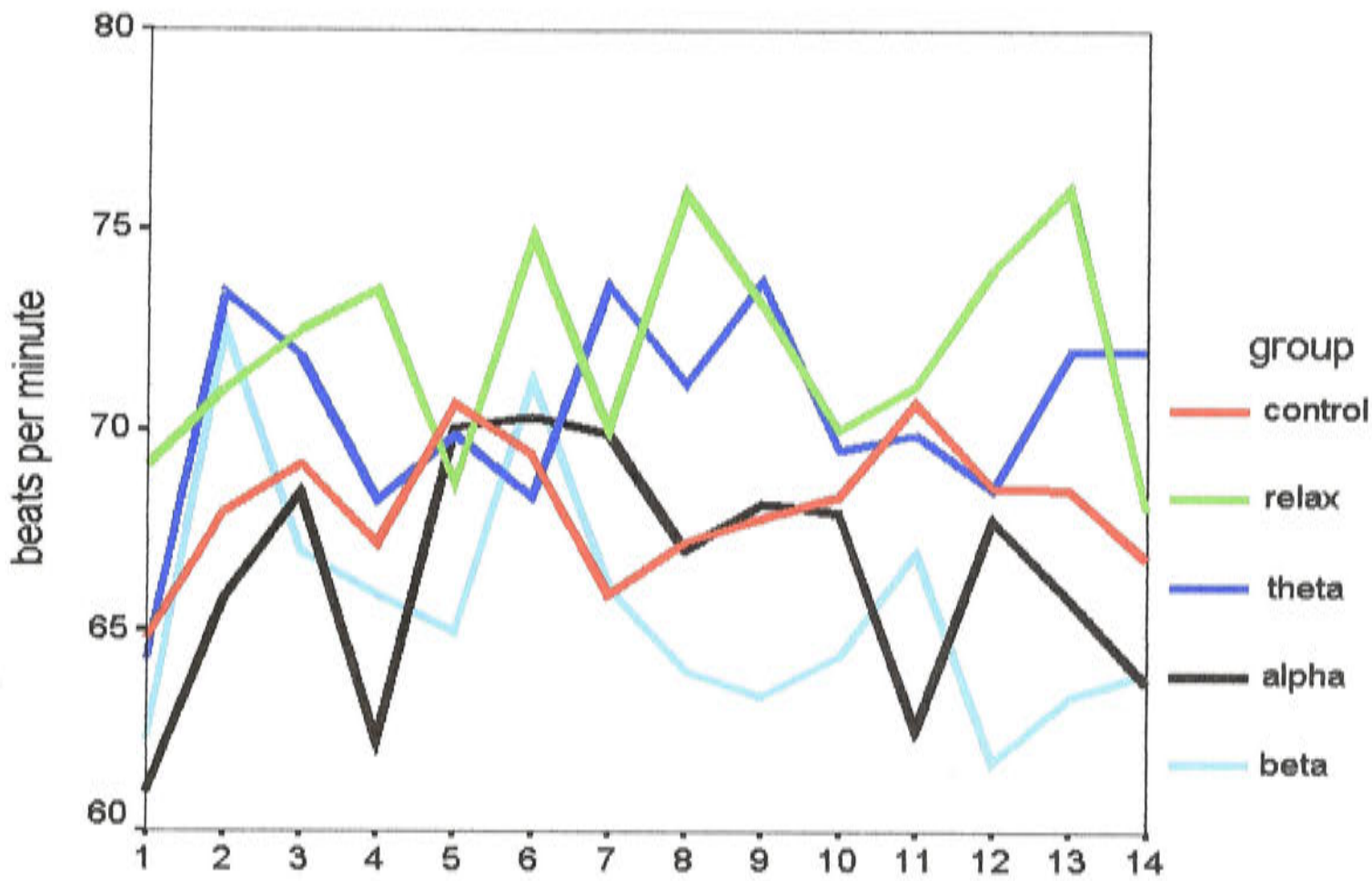



Figure 2.1 Mean heart rate at baseline (1), sessions 2-13, & follow-up (14), (n=30).

It was predicted that the theta, alpha and relaxation groups would demonstrate greater reductions in arousal, as indicated by lower heart rate and skin conductance levels, than the beta or control conditions. As can be seen in Figure 2.2 skin conductance was variable across the fourteen sessions, with the beta group showing higher skin conductance levels than the other groups. Despite this, there was no main effect for session, $F(13,13) = 1.60$, $p > .05$, $\eta^2 = .62$, and contrary to predictions, neither heart rate nor skin conductance levels changed over the sessions as a function of group membership as evidenced by non-significant session by group

interactions for both heart rate, $F(52,56) = 1.02$, $p > .05$; $\eta^2 = .48$, and skin conductance, $F(52,64) = 0.90$, $p > .05$, $\eta^2 = .42$.

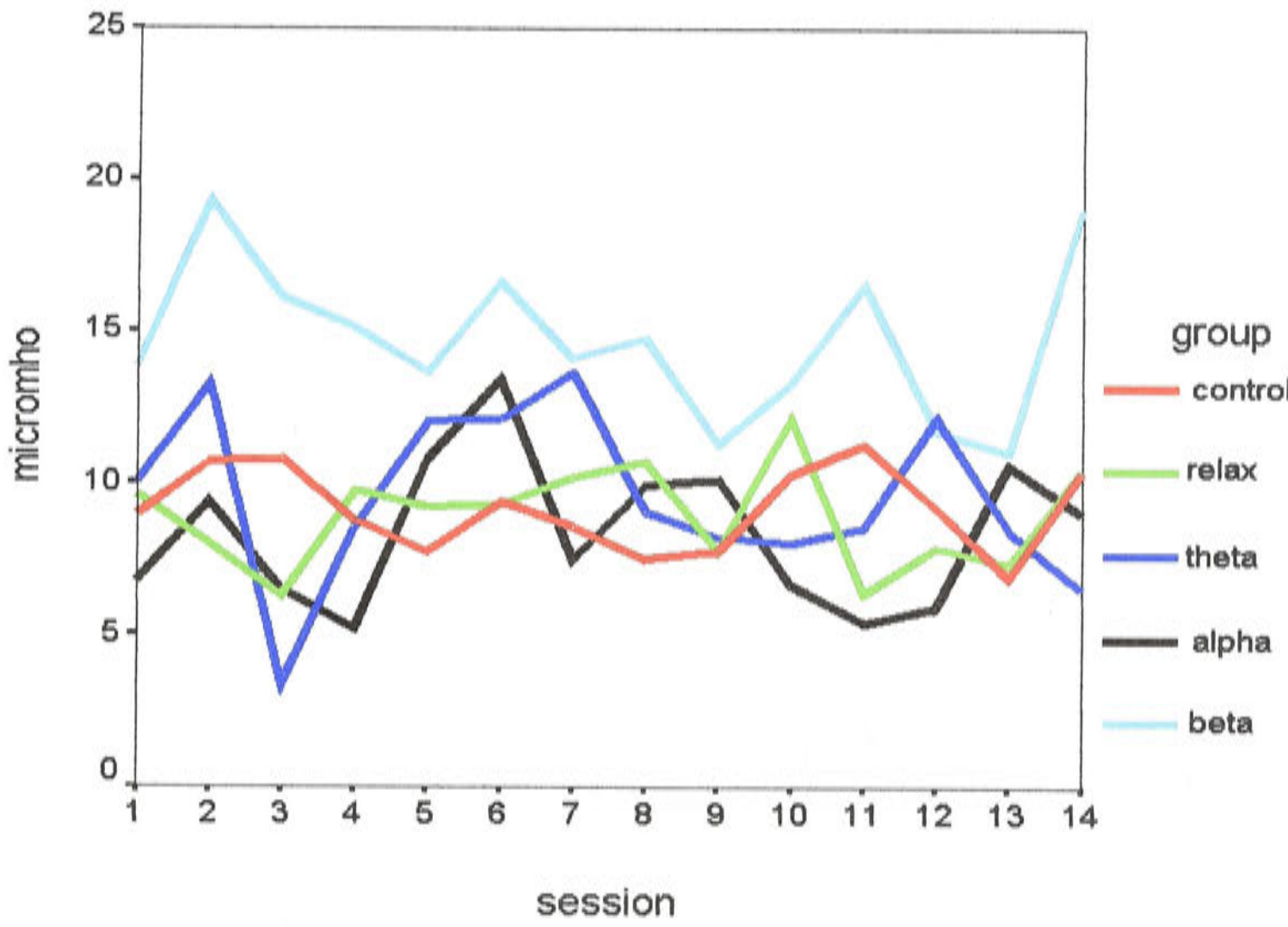



Figure 2.2: Mean skin conductance at baseline (1), sessions 2-13, & follow-up (14), (n=30).

**2.9.3 Subjective relaxation**: It was also anticipated that the theta, alpha and relaxation groups would report higher levels of subjective relaxation than the beta or control conditions during experimental trials. This hypothesis was tested using a two way MANOVA with groups (control, relaxation, theta, alpha and beta) as the between groups variable and session (1-14) as the within groups variable. This prediction was not supported (see Figure 2.3). While there was an increase in rated relaxation across sessions, as indicated by a significant main effect for session, $F(13,13) = 3.13$, $p < .05$, $\eta^2 = .76$, this effect was not dependent on group

membership with a non-significant group by session interaction obtained, $F(52, 64) = 0.54$, $p > .05$, $\eta^2 = .30$. There was a significant main effect for group, however, $F(4, 25) = 3.15$, $p < .05$, $\eta^2 = .33$. This was because the alpha group consistently reported higher subjective relaxation than any of the other groups across sessions.

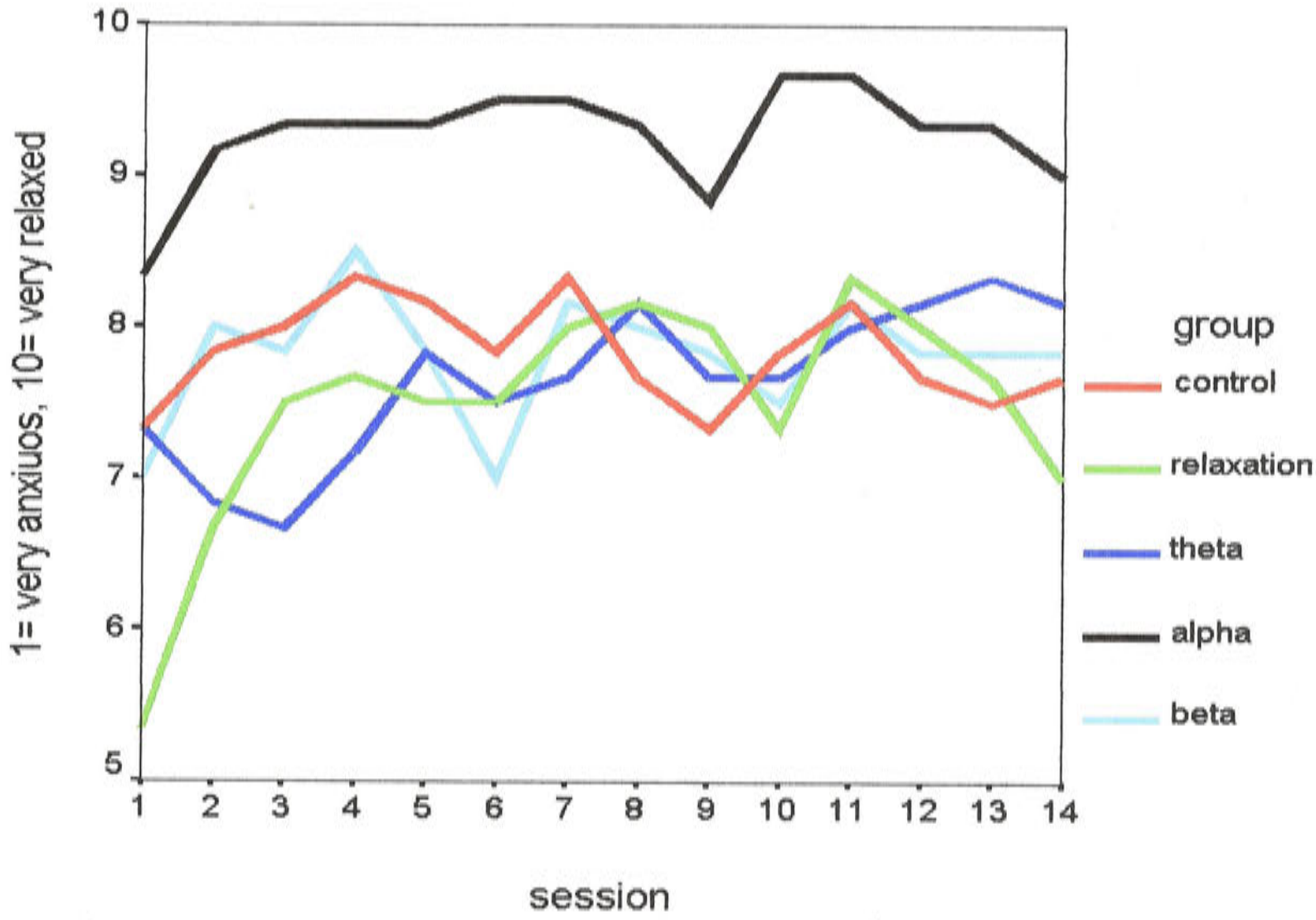



Figure 2.3 Mean subjective relaxation at baseline (1), sessions 2-13, & follow-up (14), (n=30).

**2.9.4 Mood (PANAS):** As can be seen in figures 2.4 and 2.5 changes in affect were evident for both positive and negative affect across the fourteen sessions, but not always in the intended directions. It was hypothesised that the theta, alpha and relaxation groups would report higher levels of positive affect and lower levels of negative affect than the beta or control groups during the experimental phase.

Separate three way MANOVA's were performed to test these predictions with group (control vs. relaxation vs. theta vs. alpha vs. beta) as the between groups variable, and session (1-14) and scale items (10 positive items or 10 negative items) as the within groups variables, for both positive affect and negative affect.

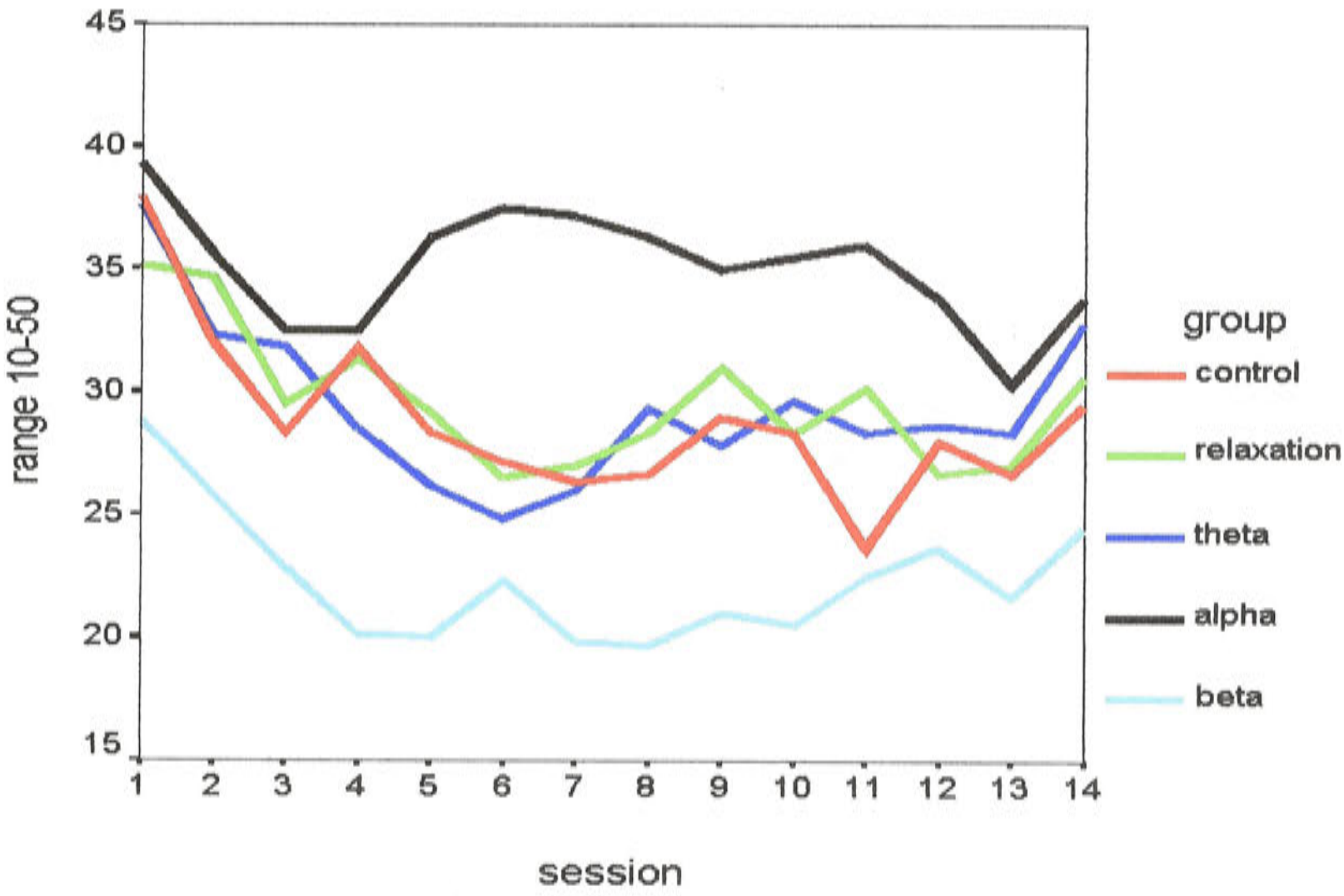



Figure 2.4 Mean positive affect at baseline (1), sessions 2-13, & follow-up (14), (n=30).

Contrary to predictions positive affect generally declined over the fourteen sessions. This was statistically significant with a strong main effect for session revealed, $F(13,13) = 6.24$, $p < .05$, $\eta^2 = .86$. Exploration of this session effect revealed positive affect, regardless of group membership, was consistently lower at every session in comparison to baseline, $F(1,25) = 1.3$ to $69.1$, $p < .05$. There was, however, a trend in expected directions. For example, there was an increase in

positive affect for the alpha group after session 4, and a sustained decrease in positive affect for the beta group throughout their therapy sessions. These observed trends, however, were not significant with a non-significant main effect for group, $F(4,25) = 1.64$, $p > .05$, $\eta^2 = .19$, and no session by group interaction, $F(52, 64) = 1.04$, $p > .05$, $\eta^2 = .46$, obtained.

There was a main effect for items, $F(9,17) = 9.3$, $p < .001$, $\eta^2 = .83$, signifying that certain items, such as 'interested', 'enthusiastic', 'inspired', 'proud', 'alert', 'active' and 'determined' were endorsed more than others, but there was no item by group interaction, which indicated that AVS or relaxation did not elicit particular mood states over and above the other conditions, $F(36,80) = 0.75$, $p > .05$, $\eta^2 = .25$.

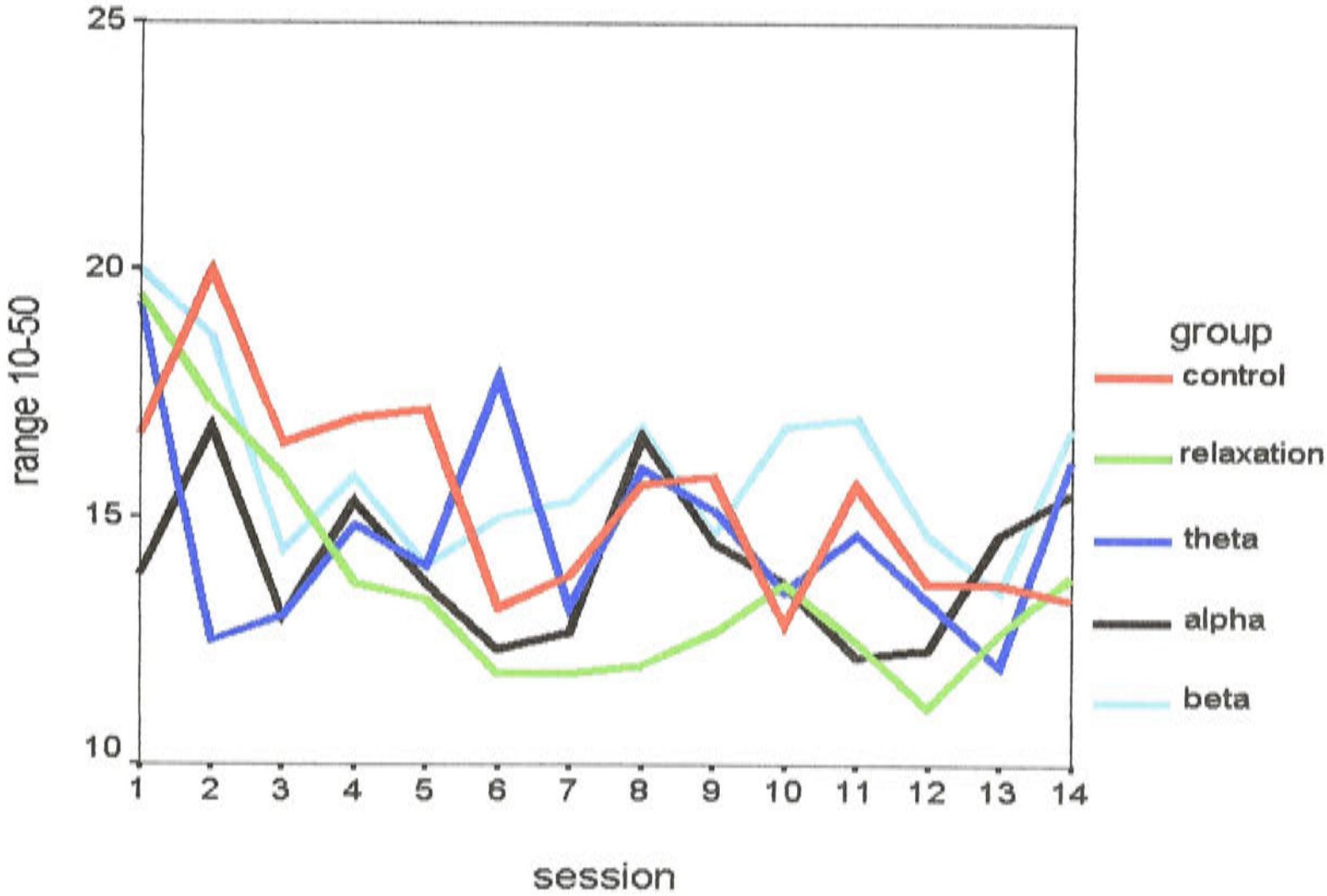



Figure 2.5 Mean negative affect at baseline (1), sessions 2-13, & follow-up (14), (n=30).

As can be seen in Figure 2.5 negative affect declined for all groups across the fourteen sessions. This was supported with a significant main effect for session, $F(13,13) = 2.61$, $p < .05$, $\eta^2 = .72$. Exploration of this effect showed that negative affect was consistently lower than baseline levels for every session except session two, $F(1,25) = 5.80$ to 33, $p < .05$. It was anticipated, however, that the theta, alpha and relaxation groups would show greater declines in negative affect than the beta and control groups during the active experimental trials. This was not supported, with only a small to moderate non-significant session by group interaction effect revealed, $F(52,64) = 1.05$, $p > .05$, $\eta^2 = .46$, and no main effect for group, $F(4,25) = 0.41$, $p > .05$, $\eta^2 = .06$. There was a significant main effect for item, however, $F(9, 17) = 7.99$, $p < .05$, $\eta^2 = .80$, indicating that participants gave greater endorsement to adjectives, such as 'distressed', 'upset', 'irritable' and 'nervous', but this was not dependent on group membership, $F(36,80) = 0.84$, $p > .05$, $\eta^2 = .27$.

**2.9.5 Psychological status (SCL-90-R):** As a non-clinical sample was used in this study it was anticipated that minimal psychopathology would be present and all participants would be within 2 standard deviations from the norm. As reported above 25% of the sample had depression, anxiety or symptom severity T-scores 1 standard deviation or more above the norm. Nevertheless, all participants were within 2 standard deviations of the norm, as expected. Despite the limited variability across scores, it was anticipated that depression, anxiety and symptom severity scores, plus negative automatic thinking, would decrease more for the theta, alpha and relaxation groups, because of enhanced relaxation and mood responses, in comparison to the beta and control groups. Congruent with this hypothesis, positive automatic thinking was predicted to increase more for the theta, alpha, and relaxation

groups over and above that of the beta and control conditions. These predictions were assessed using separate 2-way repeated measures MANOVA's with group (control vs. relaxation vs. theta vs. alpha vs. beta) as the between groups variable and session (baseline vs. follow-up) as the within groups variable, for each variable. There were no group differences in these variables at baseline.

**2.9.6 Depression**: In keeping with the selection of a 'normal' sample it can be seen in Table 2.1, that depression levels were low and within 1SD of the norm, for all groups at baseline. Indeed, the mean T-score for the alpha group was below the norm. There was a significant main effect for session, $F(1,25) = 10.86$, $p < .05$, $\eta^2 = .30$, with a decrease in depression scores at follow-up for all but the alpha group. The predicted pattern of the theta, alpha and relaxation groups showing greater reductions in depression level than the beta and control conditions at follow-up was only partially demonstrated with the theta and relaxation groups showing greater change over time compared to the other groups. This effect, however, was not significant as indicated by a non-significant session by group interaction, $F(4,25) = 1.17$, $p > .05$, $\eta^2 = .16$.

Table 2.1 Mean depression (SCL-90-R) T-Scores at baseline and follow-up.

| | Baseline | | Follow-up | |
|---|---|---|---|---|
| Group | Mean | SD | Mean | SD |
| Control | 56.8 | 8.6 | 50.6 | 12.4 |
| Relaxation | 55.5 | 9.6 | 48.6 | 7.0 |
| Theta | 52.3 | 6.1 | 44.3 | 6.8 |
| Alpha | 47.5 | 9.7 | 48.8 | 7.3 |
| Beta | 55.6 | 9.6 | 50.3 | 7.5 |

n=30

Depression correlated strongly with anxiety at both baseline and follow-up, $r = .60$ to .70, $p < .05$. It also correlated strongly in expected directions with symptom severity, $r = .80$, $p < .05$; negative affect, $r = .65$, $p < .05$; and negative automatic thoughts, $r = .70$, $p < .05$; and moderately with hassles, $r = .57$, $p < .05$; positive affect, r -.43, $p < .05$; and positive automatic thoughts, $r = -.36$, $p < .05$, as anticipated (see Appendix D, 'Ranges of Pearson's correlation coefficients for Study 1 variables').

**2.9.7 Anxiety**: The results for anxiety paralleled those for depression in that baseline T-scores were within 1SD of the norm with the alpha group exhibiting a mean T-score below the norm (see Table 2.2). There were no differences between groups at baseline as indicated by a non-significant main effect for group, $F(4,25) = .96$, $p > .05$, $\eta^2 = .13$. Anxiety levels declined from baseline to follow-up for all groups except the alpha group as indicated by a significant main effect for session, $F(1,25) = 11.10$, $p < .05$, $\eta^2 = .30$. While the theta, relaxation and beta groups demonstrated greater reductions in anxiety than the alpha or control conditions at follow-up, this was not significant, $F(4,25) = 1.94$, $p > .05$, $\eta^2 = .24$. Hence, the expected pattern of greater reductions in anxiety for the relaxation, theta, and alpha groups relative to the beta and control conditions was not supported.

Table 2.2 Mean anxiety (SCL-90-R) T-Scores at baseline and follow-up.

| | Baseline | | Follow-up | |
|---|---|---|---|---|
| Group | Mean | SD | Mean | SD |
| Control | 53.3 | 10.5 | 52.8 | 11.5 |
| Relaxation | 54.6 | 7.0 | 49.5 | 6.7 |
| Theta | 55.5 | 8.3 | 46.5 | 9.3 |
| Alpha | 45.3 | 10.7 | 45.5 | 9.6 |
| Beta | 57.8 | 8.3 | 49.8 | 7.8 |

n=30

Anxiety correlated strongly with negative affect and negative automatic thoughts across sessions with a range of Pearson's correlation coefficients of $r = .40$ to $.72$, $p < .05$ and $r = .25$, $p > .05$ to $r = .52$, $p < .05$, respectively. Smaller correlations were seen between anxiety and positive affect $r = -.33$, $p > .05$, positive automatic thoughts, $r = -.25$, $p > .05$, and hassles $r = .39$, $p < .05$.

**2.9.8 Symptom severity (Global Severity Index):** As can be seen in Table 2.3, symptom severity decreased from baseline to follow-up as evidenced by a significant main effect for session, $F(1,25) = 26.44$, $p < .05$, $\eta^2 = .51$. This decrease was not dependent on group membership, however, with a non-significant session by group interaction revealed, $F(4,25) = 0.99$, $p > .05$, $\eta^2 = .14$. While the anticipated outcome of the theta, alpha, and relaxation groups showing greater reductions in symptom severity than the beta and control conditions, was not statistically supported, the alpha and theta groups, but not the relaxation group, did demonstrate greater reductions in symptom severity than the control condition. There was no main effect for group, $F(4,25) = 1.05$, $p > .05$, $\eta^2 = .14$.

Table 2.3 Mean global severity index T-scores (SCL-90-R) at baseline and follow-up.

| | Baseline | | Follow-up | |
|---|---|---|---|---|
| Group | Mean | SD | Mean | SD |
| Control | 56.0 | 13.2 | 53.1 | 14.0 |
| Relaxation | 56.5 | 8.4 | 51.5 | 6.9 |
| Theta | 53.3 | 9.1 | 43.3 | 8.5 |
| Alpha | 49.3 | 7.7 | 43.5 | 11.6 |
| Beta | 56.2 | 4.9 | 49.8 | 2.3 |

n=30

**2.9.9 Positive automatic thoughts:** With reference to table 2.4, contrary to predictions, but commensurate with the trend for positive affect, positive thinking decreased over the experimental period, with a significant main effect for session observed, $F(1,25) = 10.70$, $p < .05$, $\eta^2 = .30$. There was no session by group interaction, however, $F(4,25) = 0.29$, $p > .05$, $\eta^2 = .04$, and no main effect for group, $F(4,25) = 1.35$, $p > .05$, $\eta^2 = .12$.

Table 2.4 Mean positive thoughts (ATQ-P) at baseline and follow-up.

| | Baseline | | Follow-up | |
|---|---|---|---|---|
| Group | Mean | SD | Mean | SD |
| Control | 102.5 | 23.2 | 83.3 | 21.7 |
| Relaxation | 82.8 | 29.3 | 72.5 | 34.8 |
| Theta | 91.8 | 29.7 | 76.2 | 34.5 |
| Alpha | 114.3 | 26.8 | 100.5 | 34.8 |
| Beta | 77.6 | 25.0 | 71.0 | 36.9 |

n=30

**2.8.10 Negative automatic thoughts:** As can be seen in Table 2.5, negative automatic thoughts decreased over the course of the study for all groups. This was supported with a significant main effect for session, $F(1,25) = 12.74$, $p < .05$, $\eta^2 = .34$. The theta, alpha, and relaxation groups did not show greater decreases in negative thinking than the beta or control conditions as indicated by a non-significant session by group interaction, $F(4,25) = .40$, $p > .05$, $\eta^2 = .06$.

Table 2.5 Mean negative automatic thoughts (ATQ) at baseline and follow-up.

| | Baseline | | Follow-up | |
|---|---|---|---|---|
| Group | Mean | SD | Mean | SD |
| Control | 50.1 | 11.6 | 41.2 | 15.3 |
| Relaxation | 43.6 | 10.3 | 34.6 | 3.5 |
| Theta | 42.6 | 14.8 | 34.0 | 3.5 |
| Alpha | 38.8 | 8.4 | 36.6 | 5.8 |
| Beta | 47.8 | 8.8 | 39.6 | 8.0 |

n=30

**2.9.11 Electroencephalography (EEG)**: EEG data were assessed prior to hypotheses testing to ensure that expected patterns of EEG amplitude were present. A four-way MANOVA with group (control vs. relaxation vs. theta vs. alpha vs. beta) as the between groups variable, and session (1-14), site (Fp1-F3, Fp2-F4, P3-O1, P4-O2), and band (theta, alpha, and beta) as the within groups variables was used to assess patterns in the EEG across the scalp and across sampled bandwidths. A main effect for band was revealed, $F(2,18) = 97.42$, $p < .05$, $\eta^2 = .91$, showing the expected pattern of higher amplitude in lower frequencies (4-6Hz and 12-14Hz) and lower amplitudes in the 21-23Hz bandwidth. Also of relevance was the observed decrease in EEG amplitude across the scalp with lower amplitudes observed at the two frontal sites (Fp1-F3, Fp2-F4) and higher amplitudes in the two posterior leads (P3-01, P4-02) (Niedermeyer & Lopes Da Silva, 1993). This was supported by a significant main effect for site, $F(3,17) = 9.45$, $p < .05$, $\eta^2 = .62$.

To test the hypothesis that there would be an increase in EEG amplitude at the frequency of AVS, separate two-way MANOVA's, with group (control vs. relaxation vs. theta vs. alpha vs. beta) as the between groups variable, and session (1-14) as the within groups variable, were computed for each bandwidth. Because of missing data in some participants' EEG, due to either equipment failure or removal due to excessive artifact, a more complete MANOVA model could not be used. In order to assess the amount of entrainment across the occiput, the two occipital sites (P3-01 and P4-02) were summed to obtain a single occipital measure.

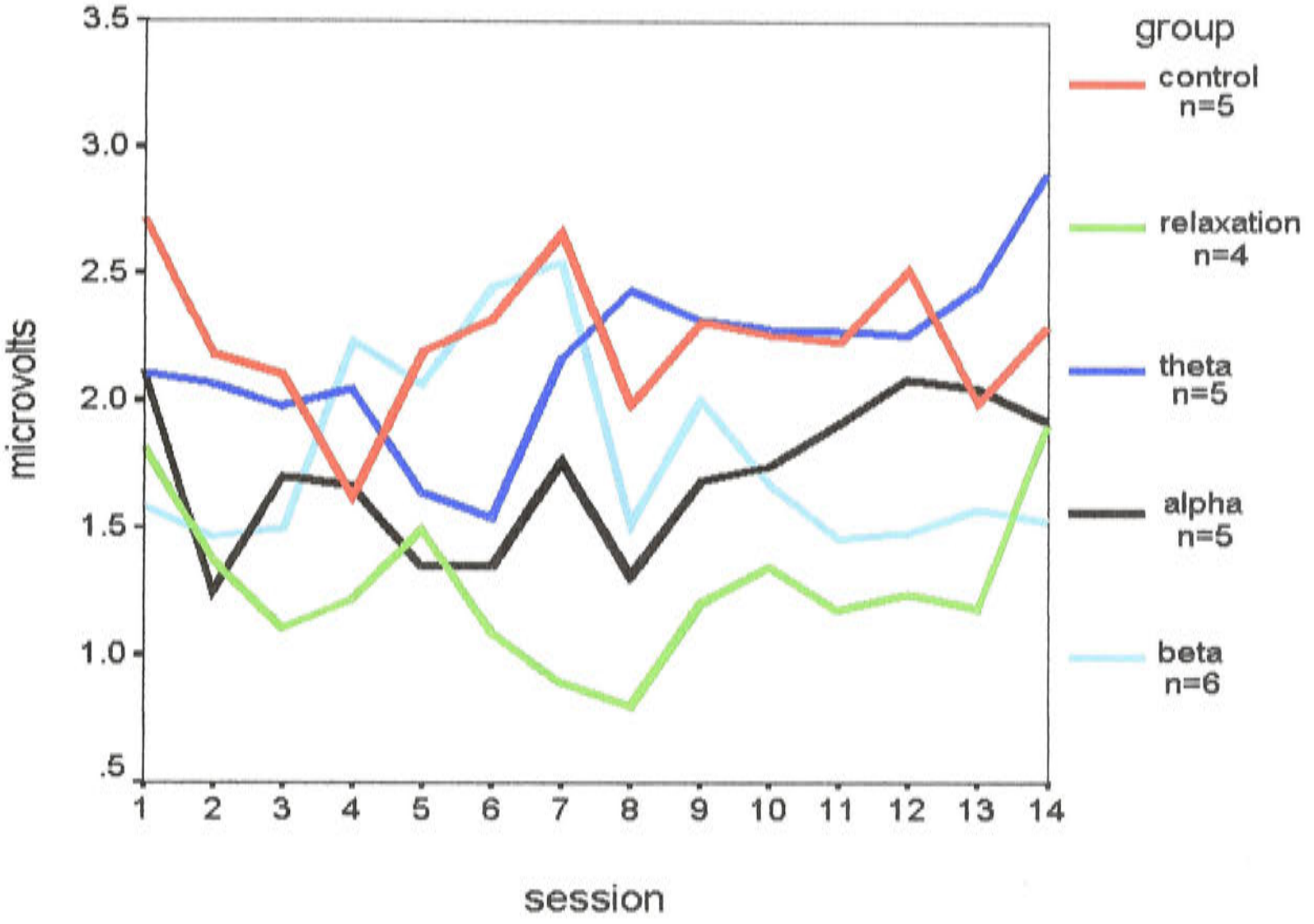



Figure 2.6 Mean amplitude in occipital 4-6Hz at baseline (1), sessions 2-13, & follow-up (14).

As can be seen in Figure 2.6, there appears to be a decrease in 4-6Hz for the relaxation and alpha groups across experimental sessions with a return to baseline levels at follow-up. The control, and theta groups appear to be relatively unchanged over time, while the beta group exhibits an increase then a decrease in this bandwidth. It was anticipated that the theta group would show increases in 4-6Hz in their EEG in response to the 5Hz AVS stimulation they were receiving. This was not found. There was no main effect for session, $F(13,8) = 2.30$, $p > .05$, $\eta^2 = .79$, and no session by group interaction, $F(52,44) = .91$, $p > .05$, $\eta^2 = .51$. In addition, there were no differences between groups as indicated by a non-significant main effect for groups, $F(4,20) = 2.07$, $p > .05$, $\eta^2 = .29$.

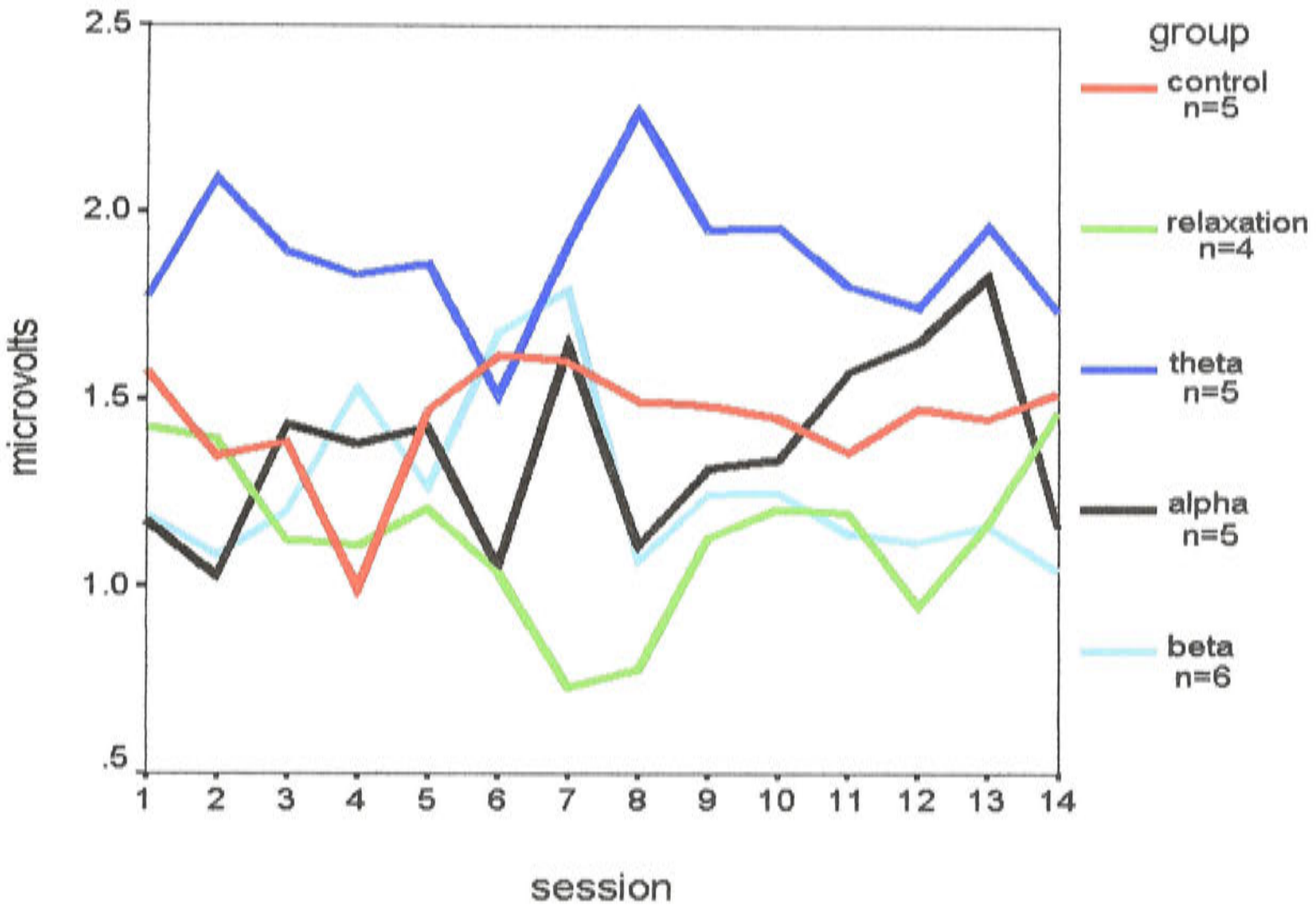



Figure 2.7 Mean amplitude in occipital 12-14Hz at baseline (1), sessions 2-13, & follow-up (14).

Similarly, for the alpha group, who received 13Hz AVS, there was little evidence of entrainment in the 12-14Hz bandwidth. As can be seen in Figure 2.7 there was a small increase in amplitude for the alpha group after session two, which was sustained until follow-up. Contrary to predictions, however, the theta group showed the highest amplitude of 12-14Hz in their EEG, which was sustained across sessions. These observed patterns were not statistically significant with no group effect present, $F(4,20) = 1.35$, $p > .05$, $\eta^2 = .21$, and no session by group interaction, $F(52,44) = 0.83$, $p > .05$, $\eta^2 = .50$.

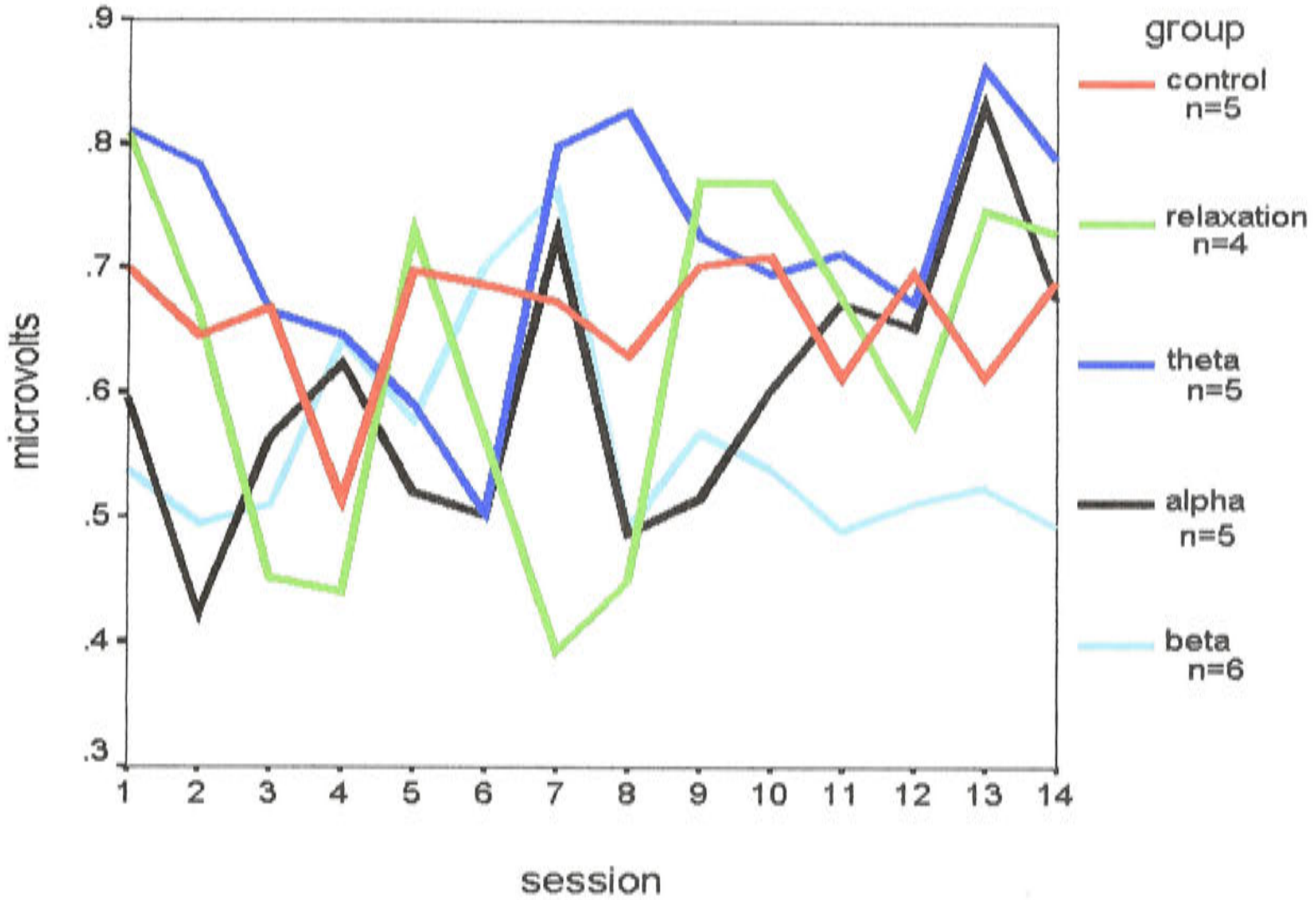



Figure 2.8 Mean amplitude in occipital 21-23Hz at baseline (1), sessions 2-13, & follow-up (14).

Examination of Figure 2.8 shows those participants receiving 22Hz AVS (beta group) displayed a small increase in 21-23Hz amplitude after session four only to fall again to baseline levels by session eight. However, this small change is indistinguishable from the background of activity in other groups whereby amplitude in the 21-23Hz bandwidth was highly variable, within a restricted range, for all groups. There was no group effect revealed, $F(4,21) = 0.59$, $p > .05$, $\eta^2 = .10$, and no session by group interaction, $F(52,48) = 1.13$, $p > .05$, $\eta^2 = .55$. Independent of group membership, amplitude in 21-23Hz did not vary significantly across sessions, $F(13,9) = 2.70$, $p > .05$, $\eta^2 = .80$. Thus, there was no evidence to support the proposal that entrainment effects would be evident during AVS exposure and remain at follow-up.

To assess inter correlations between EEG and psychological variables, three global EEG measures, 'global theta', 'global alpha', and 'global beta', were computed by summing across all sites for each respective bandwidth. There was little consistent relationship between EEG and psychological variables across sessions with highly variable patterns observed (see Appendix D). Positive affect tended to correlate negatively with global theta and global alpha with a large range across sessions from no relationship to significant moderate relationships such as, $r = -.40$, $p < .05$ with global theta, and $r = -.38$, $p < .05$ with global alpha. A similar wide range of correlations was observed for global beta and positive affect, with a range from $r = -.02$ to $.32$, $p > .05$. Thus positive affect was associated with low theta and low alpha, but unrelated to beta. Negative affect was positively related to global beta frequencies as hypothesised, $r = -.33$, $p > .05$ to $.42$, $p < .05$, but was unrelated to global theta, or global alpha, $r = -.30$ to $.30$, $p > .05$, across sessions.

EEG did vary with menstrual cycle. Pearson's correlation coefficients across sessions between menstrual cycle and global theta and global alpha ranged from no relationship to moderate inverse relationships of, $r = -.50$, $p < .05$, and $r = -.46$, $p < .05$, respectively. There was no relationship between menstrual cycle and global beta. Thus analysis indicated that as the luteal phase of the menstrual cycle progressed, global theta and alpha tended to decrease. Converse to expectations, as women approached the end of their cycle, positive affect increased, $r = .55$, $p < .05$, while negative affect decreased, $r = -.41$, $p < .05$.

Pearson's correlation coefficients between heart rate and skin conductance ranged from non significant to moderate, $r = -.04$, $p > .05$, to $r = .52$, $p < .05$ across sessions. Both heart rate and skin conductance correlated with global EEG variables ranging from very small non-significant correlations to moderate correlations. For example, heart rate correlated with global theta, $r = -.40$, $p < .05$; global alpha, $r = .38$, $p < .05$, and global beta, $r = .50$, $p < .05$. Similarly, a range of small non significant to moderate correlations were found between skin conductance and global theta, $r = .58$, $p < .05$, global alpha, $r = .43$, $p < .05$, and global beta, $r = .46$, $p < .05$. Both heart rate and skin conductance were unrelated to positive affect, but moderately related to negative affect, $r = .37$ and .39, $p < .05$, respectively.

## 2.10 Discussion of results

The main aim of this study was to establish if brainwave entrainment was better than autogenic relaxation at inducing both physiological and self-reported reductions in arousal. It also sought to ascertain if various frequencies of AVS impacted on brain functioning and differentially affected relaxation and mood. Overall, the results do not support the contention that brainwave entrainment is superior to traditional relaxation methods. Analysis revealed only small to moderate non-significant session by group interaction effects, essentially indicating that the various experimental manipulations had little differential effect on mood and arousal.

In line with predictions, relaxation responses were elicited for every group with demonstrated deceases in heart rate and skin conductance at each session. Congruent with these findings, self reported relaxation increased across the study period for all groups. There was no support for the prediction, however, that AVS and relaxation

would enhance well-being, and elicit greater relaxation responses over and above sitting quietly with eyes closed, as only small differences were observed between AVS, relaxation and control. Given these general relaxation effects during sessions, it may be expected that carry over or training effects, indicating a homeostatic change in autonomic functioning towards more parasympathetic activation, may have been found for all groups. This was not the case. Heart rate and skin conductance levels, while somewhat variable across sessions, remained relatively unchanged at follow-up for all groups.

Evidence, however, that AVS of varying frequencies did behave in predicted directions can be found in the pattern of non-significant effects across certain variables. For example, the beta group, who received 22Hz AVS, was expected to demonstrate the least amount of physiological relaxation during stimulation. As can be seen in Figure 2.2, the beta group showed a consistent and sustained increase in skin conductance throughout the experimental phase suggesting higher sympathetic arousal than the other groups. While this disparity was not significantly different from the other groups, it was in the anticipated direction and possibly because of the different AVS frequency they were receiving. In support of this, the observed group by session interaction effect for skin conductance was quite moderate, $\eta^2 = .42$, but with a reduced observed power of .80 reported. An experiment with df1= 52 and df2=64, a confidence level of 90% and power of .90, should be able to detect an effect of $r^2 = .33$ or greater (Smithson, 2003). Some caution needs to be exercised, however. Given the large df1 relative to df2, SPSS has a tendency to over estimate the true effect size and observed power. Nevertheless, it is possible that this effect size may have been detected had the experiment had sufficient power.

Further to this point, the observed increase in skin conductance for the beta group was not accompanied by a similar increase in heart rate as might be expected. This may suggest that the increase seen in skin conductance for the beta group was not an increase in arousal per se. In this study, however, the relationship between skin conductance and heart rate was quite variable. During some sessions there was no relationship and at other sessions, a moderate relationship was present, $r = .52$, $p < .05$. This dissociation between physiological markers is not uncommon. In Claridge's (1967) discussion of 'specificity' he points out that different people can exhibit hyperarousal via different physiological modalities, for example, as an increase in blood pressure for one, or an increase in skin conductance for another, but not all physiological variables react in concert to an arousing stimulus.

Further trends in predicted directions were found. For example, the alpha group consistently reported higher levels of subjective relaxation than the other groups across sessions (see Figure 2.3). Unfortunately, this difference was statistically significant at baseline, but not for subsequent sessions. The effect size for the session by group interaction for this variable was small to moderate, $\eta^2 = .30$, but the observed power was extremely low, .5. As previously discussed, an experiment with adequate power, for example .90, and large degrees of freedom (52,64), is capable of detecting an effect size of $r^2 = .33$. Given this, the observed effect may not have been large enough to reach significance even if the power of the study was optimal. Therefore, the evidence in the current study that alpha entrainment induced higher levels of subjective well-being is rather tenuous.

A further aim of the study was to see if entrainment responses could be observed in the EEG at the particular AVS frequencies used. Statistical analysis revealed little evidence of entrainment responses such as increases in EEG amplitude at frequencies of stimulation. This may have been because the entrainment frequencies used in the study were not within the dominant frequency range of 8-12Hz where entrainment responses are most likely to occur (Silberstein, 1995a; Toman, 1941). According to Pockberger (1985) response to light flicker is positively correlated with level of depression. This study was performed on a non-clinical sample with the majority of participants within one standard deviation of the norm. This restricted range in depression level may explain why evidence for entrainment was not found. To fully explore frequency following and its relationship to mood, future research on a clinically depressed sample could use 10Hz photic stimulation to assess whether participants respond to photic stimulation or not, and additionally, to ascertain if recovery from depression is associated with an attenuation of frequency following as purported by Pockberger (1985).

To assess frequency following responses more thoroughly average EEG amplitude was computed across experimental trials (sessions 2-13) for each bandwidth, 4-6Hz, 12-14Hz, and 21-23Hz, and compared to baseline measures. Eight participants showed increases, not only at the bandwidth of their particular AVS stimulus, but also consistently across the other bandwidths investigated. In the 4-6Hz bandwidth, five participants from the theta group, three participants from the alpha group, five participants from the beta group, and only one participant from each of the control and relaxation groups displayed increases in EEG amplitude at this frequency. Essentially, only those in the active AVS groups demonstrated increased EEG

amplitude within this bandwidth. Those receiving 13 or 22Hz AVS who showed increases within the theta bandwidth, were possibly displaying sub-harmonic effects in response to their respective frequencies of stimulation. For example, sub-harmonic responses in the theta bandwidth to 13Hz and 22Hz AVS would be seen at 6.5 and 5.5Hz respectively.

For the 12-14Hz bandwidth, a similar picture emerges. Four participants from each of the active AVS groups, alpha, theta, and beta, showed increases in EEG amplitude at this frequency across experimental trials, while only two from the control group and none from the relaxation group showed increases in EEG amplitude. Similarly, for the 21-23Hz bandwidth, four participants from the beta group, three from the alpha group, two from the theta group, two from the relaxation group and three from the control group showed increases in EEG amplitude in this bandwidth.

It appears therefore, that more participants in the active AVS conditions displayed increases in EEG amplitude, not only at their particular frequency of AVS, but across the other EEG bandwidths of interest. Omnibus MANOVA, however, did not reveal these effects, because of small to moderate effects sizes, and insufficient power in the experimental design. Moderate session by group interactions were revealed, $\eta^2 =$ .52, .50, and .55 for the 4-6Hz, 12-14Hz, and 21-23Hz bandwidths respectively, with power ranging between .72 to .86. An experiment with df1=52, and df2=44, with a confidence level of 90%, and power of .90, however, should be capable of revealing an effect of $r^2 = .40$ or greater (Smithson, 2003). Given that this test has a large df1 relative to df2, it is likely that SPSS has overestimated the given effect size as

previously discussed. However, it is possible, given adequate experimental power, that these interaction effects may have reached significance.

In line with predictions, negative affect declined over experimental trials, but contrary to predictions, positive affect also declined. The anticipated pattern of the alpha, theta and relaxation groups showing the greatest decreases in negative affect and greater increases in positive affect was not seen. It was, however, partially supported with the alpha group reporting greater positive affect over experimental trials and the beta group reporting less positive affect than the other groups. Relaxation appeared to have the most beneficial effect on negative affect, with greater declines observed for the relaxation group across sessions in comparison to the other groups. The effect sizes for the session by group interactions were moderate, $\eta^2 = .46$ for both positive and negative affect, but with insufficient power available to detect these, power = .80. As previously highlighted, if the power in these tests had been .90 or higher (df=52,44), these interaction effects may have been detected. Either this experiment failed to detect these small effect sizes, or they were too small in the first instance to be detected.

In the current study affect was related to EEG but not always in the directions suggested by the literature. Positive affect tended to be inversely related to theta and alpha. This finding contradicts previous research which claims that alpha is associated with positive mood states. Theta is purported to be associated with drowsiness and hypnagogic states, which some people may perceive as a negative rather than a positive experience. Negative affect, on the other hand, was unrelated to theta and alpha EEG, but positively related to beta frequencies. Beta has been

associated with anxiety states (Pizzagalli et al., 2002). In this sample, however, anxiety did not mediate negative affect as anxiety was unrelated to beta activity. Rather, some other aspect of negative affect facilitated the relationship, possibly physiological arousal, as indicated by significant positive correlations between beta, heart rate, and skin conductivity.

Of interest in this initial study, was the observed reduction in depression, anxiety, and symptom severity, in an essentially non-clinical sample. The expected trend of the theta, alpha and relaxation groups reporting greater reductions in depression and anxiety was not statistically supported. The alpha group commenced the study with depression and anxiety scores below the mean T-score of 50, and consequently were hampered by floor effects. Regression towards the mean dictates that this group may increase their scores over time purely by chance. This in fact occurred with a small trend back towards the mean for depression and little change in anxiety scores. The most benefit appeared to occur for the theta, relaxation, and beta groups who showed the greatest reductions in depression and anxiety over the course of the study, while minimal changes were observed for the control group. Session by group interactions for depression anxiety and symptom severity, however, were quite small and non significant. Nevertheless, it appears that those in the active therapy conditions experienced the greatest amount of change.

## 2.11 Summary

The main aim of this study was to ascertain if AVS was superior to traditional relaxation techniques in its ability to elicit both physiological and self reported relaxation responses. The results showed AVS to be as efficacious as relaxation in

its ability to induce relaxation responses and impact on mood. Small trends in expected directions were found. Alpha stimulation tended to make participants feel more relaxed, with increased positive affect, in comparison to relaxation, control, or the other AVS frequencies. This is consistent with previous research which reported alpha states to be associated with positive feelings, a sense of 'letting go' and increased well-being (Brown, 1970, 1971; Morse, 1993, 1994a; Nowlis & Kamiya, 1970). Beta entrainment appeared to increase arousal, to a small degree, as predicted with lower positive affect and higher skin conductance levels observed for this group. Overall, however, while small trends were present, statistical evidence showing brainwave entrainment to be superior to relaxation therapy was not found. This is consistent with Walach (1998) who found that brainwave entrainment techniques faired no better than sham (participants sat quietly with their eyes closed for up to forty minutes) in its ability to enhance well-being and produce relaxation responses. In Walach's study, however, brainwave entrainment techniques were not equivalent to electrical stimulation or sham, as they did produce more visual imagery than the other conditions.

In summary, the current study found audiovisual stimulation to be a reliable method for inducing immediate relaxation. It was found to be equally as efficacious as autogenic relaxation in its ability to produce decreases in physiological arousal and subjective relaxation, similar to that found in previous studies (Morse, 1993, 1994a; Ossebaard, 2000; Walach, 1998). In addition, AVS incorporates some of the necessary components of relaxation as described by Benson (1975); a repetitive stimulus on which to focus, and restriction of external stimuli, coupled with the added benefit of entrainment effects acting upon the cortex. In this study, however,

audiovisual stimulation, autogenic relaxation, and placebo, produced significant reductions in depression, anxiety and symptom severity, in a largely non-clinical sample, possibly augmented by relaxation responses, subtle entrainment effects, and other non-specific factors such as demand characteristics and positive expectancies.

This study, like others, found audiovisual stimulation to be equally as effective as autogenic relaxation in its ability to elicit relaxation responses (Dieter & Weinstein, 1995), but AVS differs from most relaxation therapies, because it is a passive procedure which induces relaxation responses with minimal effort. In contrast, relaxation techniques, require skill, effort, concentration, and practice, which requires motivation, in order to create the desired effects. Therefore, it is hypothesised, that brainwave entrainment may have greater clinical utility in the long-term treatment of arousal disorders, such as depression, because of its ease of use, and ability to quickly induce relaxation responses, it may overcome problems with compliance and motivation which are common in depression. The current study revealed small to moderate effect sizes using a non-clinical sample, therefore, stronger treatment effects are anticipated in the next study, which will use a sleep disturbed, clinically depressed sample, who will have a greater capacity to show improvement.

In conclusion, therefore, brainwave entrainment techniques have the potential to be more effective than traditional relaxation therapies in the treatment of stress-related disorders, such as depression for several reasons. They have proven anxiolytic qualities, plus the ability to entrain the cortex into more conducive brainwave states. Finally, mind machines require no prior training, are easy to use, novel and popular

devices. They offer a coping strategy that can enhance treatment compliance and maximise treatment outcomes in often difficult to treat depressed individuals, who have difficulty applying adequate coping skills when dysphoric, sleep disturbed, and deficient in motivation.

# CHAPTER 3

## 'Depression defined as a stress-related disorder'

"I like living. I have sometimes been wildly, despairingly, acutely miserable, racked with sorrow, but through it all I still know that just to be alive is a grand thing."
- Agatha Christie (1890-1976)

### 3.1 Introduction

Depression is on the increase in industrialised and developing countries and is the biggest cause of morbidity, functional impairment, and loss of productivity with a prediction of increasing social costs in the future (Murray & Lopez, 1997; Murray & Lopez, 1996b). Agatha Christie poignantly describes the experience of depression. Pervasive sadness and loss of interest and pleasure in activities, most days, for at least two weeks are the main defining features of an episode of Major Depressive Disorder (MDD) as outlined by the American Psychiatric Association (APA), 1995. In order to qualify for diagnosis, MDD also includes some of the following symptoms: loss of energy and fatigue, diminished appetitive and sexual drives, and sleep disturbance; cognitive symptoms such as memory impairment, difficulty concentrating and making decisions; behavioural features such as crying, agitation or conversely, psychomotor retardation; and finally, feelings of worthlessness, guilt or self-loathing. Suicidal ideations are common and can be indicative of the severity of the disorder. Not surprisingly, MDD causes mild to severely debilitating impairments to daily functioning (American Psychiatric Association, 1995).

### 3.2 Prevalence of Major Depression

Unipolar major depressive disorder (MDD) is usually a recurring chronic disorder which engenders personal suffering and huge social expense (Andrews, 2001; Angst,

Stassen, Clayton, & Angst, 2002; Brodaty, Luscombe, Peisah, Anstey, & Andrews, 2001; Cassano & Fava, 2002; Druss, Rosenheck, & Sledge, 2000; Goldney, Fisher, Wilson, & Cheok, 2000; Herrman et al., 2002; Maj, Veltro, Pirozzi, Lobrace, & Magliano, 1992; Murray & Lopez, 1996a; Pincus & Pettit, 2001). According to the World Health Organisation report, 'The Global Burden of Disease' (1996), MDD is a major cause of disability in the world, accounting for 11% of total disability, and is projected to be the leading cause of disability in females by 2020 (Murrary & Lopez, 1996; Murray & Lopez, 1996a; Murray & Lopez, 1997). Lifetime prevalence rates for MDD in America and Western Europe range from 13.3 to 17.1% (Carta et al., 1995; Cassano & Fava, 2002). A similar picture has been found for Australia. In the '1997 National Survey of Mental Health and Wellbeing of Adults', approximately 6% of people reported suffering from an affective disorder and 9.5% reported suffering from an anxiety disorder in the 12 months preceding the survey. The survey found that women were nearly twice as likely to suffer from an anxiety or affective disorder than men (Australian Bureau of Statistics, 1998; Henderson, Andrews, & Hall, 2000). This is consistent with other research in Australia (Byrne, 1980; Goldney et al., 2000), the United Kingdom (Jenkins et al., 1997), and the United States (Coryell, Endicott, & Keller, 1992; Desai & Jann, 2000; Wu & Anthony, 2000) which also reported higher prevalence rates of MDD for women. A common finding in mental health surveys and clinical practice is the comorbidity of anxiety and depressive illness which exacerbates the burden of suffering and increases societal costs (Devanand, 2002; Keller & Hanks, 1995; Kessler & Frank, 1997; Kessler et al., 1994; Liebowitz, 1993; Sherbourne & Wells, 1997; Simon et al., 2003; Wittchen & Essau, 1993; Yerevanian, Koek, & Ramdev, 2001).

The high comorbidity of depression with anxiety, which is a disorder of arousal, reflects current thinking that depression is a stress-related disorder (Chrousos & Gold, 1992; Nestler et al., 2002). Contemporary research shows that hyper-arousal in MDD coexists with a dysregulated and over-active hypothalamic-pituitary-adrenal axis (HPA) resulting in chronically elevated glucocorticoids which have deleterious effects on brain tissue, emotions, and behaviour (Arborelius & Owens, 1999; Chrousos & Gold, 1992; Duman, Heniger, & Nestler, 1997; Everly & Benson, 1989; Gold, Drevets, Charney, & Drevets, 2002; Holsboer, 2000a; Light, Kothandapani, & Allen, 1998; Plotsky, Owens, & Nemeroff, 2000). As a direct consequence, MDD is typified by abnormalities in frontal and limbic brain structures (Drevets, 2000b, 2003; Soares & Mann, 1997) which are reflected in unique EEG patterns of pre-frontal under-activation (Davidson & Henriques, 2000; Davidson & Irwin, 1999; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Henriques & Davidson, 1991; Matousek, 1991). Sequelae of MDD include fatigue (Lavidor, Weller, & Babkoff, 2002), sleep disturbance, impairments of thinking, memory and concentration, and mood disturbance (American Psychiatric Association, 1995; Austin et al., 1999; Brunello et al., 2000; Erickson, Drevets, & Schulkinb, 2003).

### 3.3 Distinguishing between mood and depression

Mood is transient and highly variable from moment to moment. Factor analyses of mood measures consistently reveal that mood is primarily composed of two main orthogonal factors, a positive factor and a negative factor (Diener & Emmons, 1985; Watson, Clark, & Tellegen, 1988). According to Clark and Watson (1991) negative affect refers to "the extent to which a person is feeling upset or unpleasantly engaged rather than peaceful". Positive affect, on the other hand, represents "the extent to

which a person feels zest for life" (p. 321) and is often associated with pleasant events (Stone, 1981). Valence of affect has been used to distinguish between anxiety and depression (Clark & Watson, 1991; Clark, Watson, & Mineka, 1994; Gencoz, 2002; Watson, Clark, & Carey, 1988; Watson, Clark et al., 1995; Watson, Weber et al., 1995). Negative affect reflects a general level of subjective distress and is common to both anxiety and depression, while positive affect is negatively related to depression, but essentially unrelated to anxiety. Positive affect therefore effectively discriminates between depression and anxiety with depression comprised of high negative affect and low positive affect, while anxiety is exemplified by high negative affect only. Negative affect that is persistent, all-encompassing and interferes with daily functioning, becomes depression.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) categorises anxiety and depression as separate constructs (American Psychiatric Association, 1995). Depression is predominately characterised by mood disturbance while the major component of anxiety is fear. Yet despite this, and reflected in the observed correlations with positive and negative affect outlined above, anxiety and depression share affective components (negative affect or subjective distress) and are often comorbid (Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Clark & Watson, 1991; Katon & Roy-Byrne, 1991; Keller & Hanks, 1995; Mineka, Watson, & Clark, 1998; Nitschke, Heller, Imig, McDonald, & Miller, 2001; Rodney et al., 1997; Sherbourne & Wells, 1997; Stahl, 1993; Watson, Clark et al., 1995; Watson, Weber et al., 1995; Wittchen, Schuster, & Lieb, 2001; Yerevanian et al., 2001).

### 3.4 Defining depression as a disorder of dysregulated arousal

Lifetime comorbidity of anxiety with primary mood disorder has been found to be as high as 59% (Brown et al., 2001). Given this high comorbidity, and it is not surprising that contemporary models of affective illness implicate arousal systems in the pathogenesis of depression (Chrousos & Gold, 1992; Gold, Goodwin, & Chrousos, 1988; Leonard, 2002; Nestler, 1998). According to Everly and Benson (1989) a common neurological substrate underlies all stress-related disorders, including depression. Through repeated exposure to stressful stimuli the central nervous system becomes sensitised and permanently altered to be hyper-reactive to subsequent stimulation. Rich interconnectivity between the limbic system, the frontal cortex, and the locus coeruleus, means once the system has been 'charged', reverberating circuits within these systems work to maintain the heightened sensitivity with an abnormal preference for sympathetic activation (Everly & Benson, 1989; Post, 1992). In other words, the nervous system 'learns' to be chronically over-aroused and 'stressed' (Esch, Stefano, Fricchione, & Benson, 2002; Everly & Benson, 1989; van der Kolk, 2001).

### 3.5 The stress response

Exposure to a stressor, either environmental or psychological, activates two functionally connected arousal pathways, the sympathetic nervous system, and the hypothalamic-pituitary-adrenal (HPA) axis (Kvetnansky et al., 1995). Acute stress responses are facilitated by the sympathetic nervous system via the release of norepinephrine (NE) from the locus coeruleus (LC) in the brainstem (Herman & Cullinan, 1997). Norepinephrine helps to prepare the body for fight or flight. As indicated in Chapter one, sympathetic activation increases heart rate, cardiac output,

and respiratory rate; it dilates the pupils, stimulates the release of glucose from the liver, suppresses digestion, sexual activity and immune responses. Furthermore, it redirects blood flow to muscles in readiness for action (Kandel, Schwartz, & Jessell, 1991).

The appraisal of threat also activates the HPA system by the release of corticotropin releasing hormone (CRH) and vasopressin from the paraventricular nucleus (PVN) in the hypothalamus (Clark & Kaiyala, 2003). Both CRH and vasopressin in turn stimulate the anterior pituitary to release adrenocorticotropin hormone or ACTH (Akil & Morano, 1995; Plotsky et al., 2000; Tsigos & Chrousos, 2002; Vermetten & Bremner, 2002). The main effector organ of ACTH is the adrenal cortex, which releases glucocorticoids, mainly cortisol, into the blood stream. The role of cortisol is to prepare the organism for 'fight or flight' with the intensity of the response modulated by the severity of perceived threat (Kopin, 1995). Cortisol stimulates arousal, focuses attention, increases metabolism to ensure an adequate supply of energy, and inhibits functions not necessary for immediate survival, such as immune responses (Chrousos, 1995; Stratakis & Chrousos, 1995), neuronal repair (Chrousos & Gold, 1992), and vegetative behaviours such as eating, sex, and sleeping (Dunn & Berridge, 1990; Heinrichs, Menzaghi, Pich, Britton, & Koob, 1995; Vgontzas et al., 2001). Cortisol also acts, via a negative feedback system, to decrease the production of CRH and therefore limit the stress response (Kandel et al., 1991) (see Figure 3).

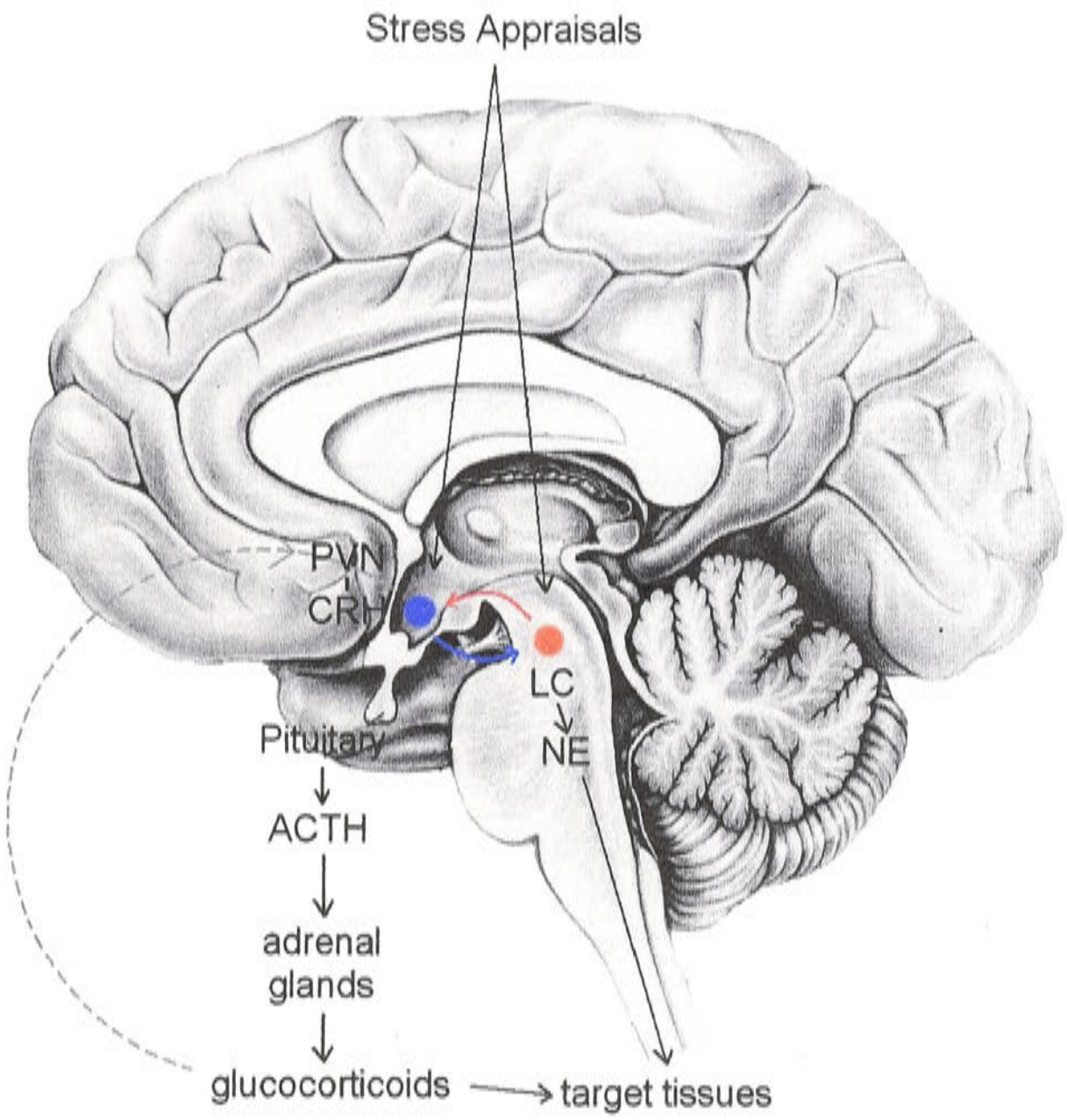



Figure 3 Central structures involved in the stress response. Appraisal of stress stimulates the release of corticotrophin releasing hormone (CRH) from the paraventricular nucleus (PVN) (blue) in the hypothalamus, & the release of norepinephrine (NE) from the locus coeruleus (LC) (red) in the brain stem. CRH stimulates the release of adrenocorticotrophin hormone (ACTH) from the pituitary, and the release of glucocorticoids from the adrenal glands, which in turn inhibit the stress response. Solid lines = stimulatory, broken lines = inhibitory. Adapted from Kandel, Schwartz & Jessell, 1991.

Activation of both arousal systems provides short term adaptation in the face of threat. Prolonged activation of the LC/NE and HPA systems, however, disrupts homeostatic balance and increases psychological distress, predisposing susceptible individuals to psychiatric pathology (Clark & Kaiyala, 2003; Mizoguchi, Ishige, Aburada, & Tabira, 2003). In MDD, arousal is chronically elevated (Chrousos & Gold, 1992; Nestler et al., 2002) and with dire consequences, such as diminished immune function (Bauer et al., 2003), sleep disturbances (Nofzinger et al., 2000),

and loss of neuronal function in frontal and limbic brain structures caused by high levels of circulating serum cortisol (Drevets, 2000b, 2001, 2003; Eskay, Chautard, Torda, Daoud, & Hamlink, 1995; Nofzinger et al., 2000).

### 3.6 Cortical anomalies present in depression

Structural and functional brain imaging has revealed that MDD is characterised by abnormalities in frontal (Harrison, 2002; Mayberg, 2003; Soares & Mann, 1997) and limbic brain structures (Drevets, 2003). Specifically, imaging studies have revealed volume reductions in the hippocampus (Bremner et al., 2000; Bremner et al., 2002; MacQueen et al., 2003; Sheline, Mittler, & Mintun, 2002; Sheline, Sanghavi, Mintun, & Gado, 1999) the cingulate (Botteron, Raichle, Drevets, Heath, & Todd, 2002), and the medial frontal cortical region (Bremner et al., 2002). Post mortem studies show decreased glial density and neuronal cell size in deep layers of the cortex in subjects with known MDD (Cotter et al., 2002; Drevets, 1999). In addition, abnormalities of blood perfusion and glucose metabolism have been found in the amygdala, thalamus, and orbital prefrontal cortex (Drevets, 1998, 2000a, 2000b, 2001, 2003; Nobler, Roose, Prohovnik, & Moeller, 2000), the insula and cingulate gyrus (Kimbrell et al., 2002), and also in the basal ganglia, and temporal lobes (Brody, Barsom, Bota, & Saxena, 2001) of depressed patients.

These structural abnormalities help to account for the deficits seen in frontal lobe functioning in depression, such as impairments in working memory, sustained attention, and other higher order frontal functions (Austin, 1995; Austin et al., 1999; Brody et al., 2001; Heller & Nischke, 1997; Henriques & Davidson, 1997; Landro, Stiles, & Sletvold, 2001; Shenal, Harrison, & Demaree, 2003). In addition, other

studies have revealed hemispheric asymmetries in depression with lower blood perfusion (MacHale et al., 2000) and reduced glucose metabolism (Martinot et al., 1990) in the left prefrontal cortex in comparison to the right. These findings are congruent with a large body of EEG research which shows underactivation of the left frontal cortex in comparison to the right frontal cortex in depressed individuals (Cook & Leuchter, 2001; Davidson, 1992; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Davidson & Henriques, 2000; Davidson & Irwin, 1999; Debener et al., 2000; Drevets, 2000b; Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991; Hinrichs & Machleidt, 1992; Knott, Mahoney, Kennedy, & Evans, 2001; Matousek, 1991; Nandrino et al., 1994; Pockberger et al., 1985; Wheeler, Davidson, & Tomarken, 1993).

## 3.7 EEG in emotion and depression

Lateralisation of brainwave activity also reflects lateralisation of emotion. Previous work in emotion by Silberman and Weingartner (1986) found that activation of the right frontal cortex was associated with the expression of negative emotion, while activation of the left frontal cortex was related to the expression of positive emotions. Davidson and colleagues further elucidated these findings and proposed that activation of the right frontal cortex is related to withdrawal from aversive stimuli and is guided by negative emotions such as disgust, fear and anxiety, while activation of the left frontal cortex reflects a propensity to approach a desired goal and is driven by positive emotions such as pleasure (Davidson & Henriques, 2000; Fox, 1991; Tomarken, Davidson, Wheeler, & Doss, 1992).

In depression, approach-withdrawal systems are disrupted because of deficits in brain functioning. Typically, the depressed person shows increased alpha activity in the left frontal cortex, indicating underactivation, in comparison to the right frontal cortex (Gotlib et al., 1998; Henriques & Davidson, 1991). Underactivation of the left frontal region robs the person of the experience of positive affect associated with seeking and obtaining pleasurable rewards (Davidson & Henriques, 2000). Given that the left frontal cortex is responsible for motivating 'approach' type behaviour towards desired goals which elicits positive feelings, it is not surprising that the core symptoms of MDD include negative affect (or attenuated positive affect), loss of pleasure and interest in activities, and a decrease in motivation to seek social contact (American Psychiatric Association, 1995). This finding of left frontal underactivity is found so consistently that Davidson proposed that it is a trait-like marker for depression which reflects a propensity towards negative affectivity and a diathesis for subsequent depression (Davidson, 1998a, 1998b, 2001; Tomarken & Keener, 1998).

Anxiety, which is often comorbid with depression, is associated with activation of right frontal (Pizzagalli et al., 2002) and right posterior (Davidson & Henriques, 2000; Heller & Nitschke, 1998; Matousek, 1991; Pozzi, Golimstock, Petracchi, García, & Starkstein, 1995) cortical regions. This may contribute to the finding of lateral asymmetries of relative right frontal hemispheric activation, especially in MDD with comorbid anxiety. High beta rhythms, which are indicative of increased cerebral activity, are also associated with arousal and are often present in recurrent depression (Matousek, 1991; Nystrom, Matousek, & Hallstrom, 1986; Pizzagalli et al., 2002), possibly reflecting a chronically dysregulated and overactive HPA and

sympathetic system. This is consistent with other research which demonstrated a positive correlation between cortisol secretion and beta EEG activity (Chapotot, Gronfier, Jouny, Muzet, & Brandenberger, 1998).

In summary, brain imaging studies attest to the damaging effects of high levels of glucocorticoids which are produced during stress responses and are often chronically elevated in MDD. Couple this with Davidson's findings of dysfunctional brainwave patterns in depression and it is apparent that MDD is characterised by very real physical changes which have emotional and behavioural ramifications and may be exacerbated and possibly triggered by repeated stress responses (Esch et al., 2002; Post, 1992). Brain imaging and EEG findings reflect pathological changes caused by chronic hyper-arousal in depression, but they fail to adequately explain the underlying mechanisms that contribute to depression and consequently inform treatment options.

## 3.8 Biological models of depression

**3.8.1 The monoamine hypothesis:** Until recently, traditional biological theories of depression have failed to acknowledge the role of stress responses in the pathogenesis of depression. The most commonly adhered to theory of depression is the monoamine hypothesis. The monoamine model purports that depression is caused by depletion of serotonin (5HT), norepinephrine (NE) (Maes & Meltzer, 1995), and possibly dopamine (DA) (Di Chiara, Loddo, & Tanda, 1999; Naranjo, Tremblay, & Busto, 2001) in the brain. The use of contemporary antidepressants is based on this model and aims to increase circulating amines in the brain by blocking pre-synaptic re-uptake or by preventing amine degradation, in an effort to increase

the availability of amines in the synaptic cleft (MIMS, 2000). The mechanisms of action, however, are complex and not merely due to increased availability of monoamines. For example, it has been shown that simply depleting monoamines does not produce dysphoria in non-depressed subjects (Arnulf et al., 2002; Booij et al., 2002). While NE and 5HT are necessary in the production of positive mood, they are not in themselves sufficient.

Further evidence of the inadequacy of the monoamine hypothesis to fully describe depression is found in its inability to explain why antidepressant treatment produces immediate increases in 5HT and NE, but therapeutic effects can take up to several weeks to occur (Nestler, 1998; Nestler et al., 2002). Rather, arousal mechanisms appear to be involved as it has been shown that effective antidepressant treatment, which increases NE and 5HT levels, subsequently dampens the overactive HPA system (Holsboer & Barden, 1996). As a consequence neurogenesis is stimulated and previously damaged cortical areas are restored to normal functioning (Duman et al., 1997).

Other research shows that serotonin is involved in arousal pathways (Dinan, 1996) and crucial to healthy sleep (Adrien, 2002). This suggests a more complex picture than that described by the monamine hypothesis and points to the need to include arousal mechanisms in models of depression (Dinan, 1996; Fuller, 1992; Maes & Meltzer, 1995; Riedel, Sobczak, Nicolson, & Honig, 2002; Vermetten & Bremner, 2002). The connection between mood and the monoamines, 5HT and NE, is not straightforward and merely increasing the amount of available neurotransmitters does not produce immediate short term improvements in mood in depressed

individuals (Holsboer, 2000a), therefore signifying that other mechanisms must be involved.

**3.8.2 The neurotrophin hypothesis:** The discovery that antidepressants increase nerve growth factors in the brain via serotonin pathways helped to explain the delayed treatment response of antidepressant medications (Altar, 1999). Research has shown that by enhancing the availability of serotonin, after some weeks, brain derived neurotrophin factor (BDNF) is increased, particularly in areas known to be vulnerable to cell damage in depression, such as the prefrontal cortex, the cingulate, and the hippocampus (Chen, Dowlasshahi, MacQueen, Wang, & Young, 2001; Duman, Malberg, Nakagawa, & D'Sa, 2000; Duman, Nakagawa, & Malberg, 2001; Shirayama, Chen, Nakagawa, Russell, & Duman, 2002). As a result, neurogenesis was restored and a reversal of depression induced atrophy was observed (Nibuya, Morinobu, & Duman, 1995; Saarelainen et al., 2003; Shirayama et al., 2002). It was assumed, therefore, that neurotrophic factors must also play a role in the pathogenesis of depression (Duman et al., 1997). It was also proposed that chronic antidepressant treatment played a protective role by preventing further damage and cell loss caused by stress responses (Nibuya et al., 1995). While the refined neurotrophic model of depression helped to explain the delayed onset of antidepressant therapy, it still did not incorporate the hyper-arousal features commonly seen in depression.

**3.8.3 The hyper-arousal hypothesis**: The hyper-arousal model of depression has turned to dysregulation of the HPA and LC/NE systems to account for accumulating evidence that depression is characterised by hyper-arousal caused by chronic over

stimulation of arousal systems (Arborelius & Owens, 1999; Holsboer, 2001; Kasckow, Baker, & Geracioti, 2001; Leonard, 2002; Light et al., 1998; Nestler et al., 2002). The hyper-arousal hypothesis is a comprehensive model which builds on the contributions of both the monoamine and the neurotrophin hypotheses to explain the mechanisms underlying depression.

It is well established that over activation of arousal systems is common in MDD. Up to 50% of depressed people show signs of over-arousal and dysregulation of the HPA system (Arborelius & Owens, 1999; de Kloet, 2002; Deuschle et al., 1997; Holsboer, 2001) with high cortisolaemia (Barden, Reul, & Holsboer, 1995; Chrousos, 1998; Galard, Catalan, Castellanos, & Gallart, 2002; Hatzinger, Hemmeter, Baumann, Brand, & Holsboer-Trachsler, 2002) and a blunted response to dexamethasone (Kasckow et al., 2001) or CRH challenge (Arborelius & Owens, 1999; Holsboer, 2001). As previously stated, chronically elevated cortisol levels damage frontal and limbic brain tissue causing cell loss and atrophy, possibly due to the downregulation of BDNF in these regions (Duman et al., 1997). As a result, the normal inhibitory feedback functions of the hippocampus are disrupted, and the ability of the hippocampus to 'switch off' stress responses is attenuated (Calfaa et al., 2003; Feldman & Weidenfeld, 1993; Mizoguchi et al., 2003). As a consequence, cortisol secretion continues unchecked. In addition, chronic activation of stress responses in depression repeatedly stimulates the LC/NE system which further exacerbates the problem of hyper-arousal with resultant damage to self limiting feedback mechanisms in this system also (Leonard, 2002).

In support of the hyper-arousal model, it has been shown that antidepressant treatment is most effective when normalisation of the HPA system has been achieved (Barden et al., 1995; Kasckow et al., 2001). Poorer treatment outcomes were found in those with histories of prolonged and recurrent episodes of depression and subsequent increased severity of HPA damage (Hatzinger et al., 2002). Because the hippocampus plays a pivotal role in regulation of the HPA system, the damage caused by high levels of cortisol prevents the negative feedback necessary to switch off the stress response once it has been activated (Duman et al., 1997; Holsboer, 2000b). It is proposed that conventional antidepressant therapy normalises the HPA system by restoring mineralcorticoid and glucocorticoid receptors in the hippocampus to normal functioning (Calfaa et al., 2003). This is made possible by increasing the availability of BDNF which in turn stimulates neurogenesis and helps to up-regulate these receptors (Bench, Frackowiak, & Dolan, 1995; Drevets et al., 2002; Holsboer, 2001).

Firmly based in the hyper-arousal model of depression, new antidepressant treatments such as CRH antagonists, aim to re-establish homeostasis by directly intervening in the stress response (Ducotteta, Griebelb, & Belzung, 2003). They work by antagonising CRH receptor sites in the anterior pituitary, thereby limiting the production of ACTH and the subsequent release of glucocorticoids which, if chronically elevated, destroys normal feedback mechanisms (Wolkowitz & Reus, 1999). Early indications are that these medications are safe to use, reduce anxiety associated with a chronically stimulated HPA system, and ameliorate dysphoria (Zobel et al., 2000). According to the hyper-arousal hypothesis, therefore, the fundamental mechanism underlying depression is dysfunction of inhibitory

mechanisms necessary for adaptive regulation of the stress response (Chrousos, 1998; Chrousos & Gold, 1992; Holsboer, 1995, 2000a).

### 3.9 Dysregulated HPA and the effects on sleep

Dysregulated stress responses, which are a core feature of MDD, also disrupt homeostatic balance and circadian regulation of sleep (Armitage, Hoffmann, & Rush, 1999; Colwell, 1995; Dinan, 1996; Linkowski et al., 1987; Riemann, Berger, & Voderholzer, 2001; Wirz-Justice, 1995). Up to 90% of depressed people complain of poor sleep with demonstrable sleep abnormalities at some time during their illness (ICSD, 1990; Okuji et al., 2002). Common sleep disturbances in MDD include delayed sleep onset, shortened latency to rapid eye movement (REM) sleep, decreased slow wave sleep (SWS), disrupted sleep, early morning wakings, and diminished sleep efficiency, with reports of feeling tired and un-refreshed after a nights sleep (Bardwell, Moore, Ancoli-Israel, & Dimsdale, 2000; Boivin, 2000; Buysse et al., 1998; De la Fuente, Bobes, Vizuete, & Mendlewicz, 2001; Kupfer et al., 1985). Indicative of the progressive nature of MDD, and the concurrence of mood and sleep anomalies (Lustberg & Reynolds, 2000), the severity of sleep disturbances increases with recurrent depressive episodes (Jindal et al., 2002; Thase et al., 1995; Vitiello, Moe, & Prinz, 2002). Furthermore, chronic sleep deprivation, which is a corollary of disrupted sleep, compromises immune system functioning, possibly due to the immunosuppressive effects of hypercortisolaemia in MDD (Rogers, Szuba, Staab, Evans, & Dinges, 2001; Savard, Laroche, Simard, Ivers, & Morin, 2003), impairs cognitive functioning (Drake et al., 2001; Smulders, Kenemans, Jonkman, & Kok, 1997), and can double the risk of death in older individuals (Dew et al., 2003).

Heightened nocturnal arousal caused by hypercortisolaemia is associated with increased severity of sleep disturbance (Madjirova, Tashev, Delchev, & Bakalova, 1995; Perlis et al., 1997). For example, nocturnal arousal decreases REM latency and increases the percentage of REM sleep at the expense of restorative slow wave sleep (Somers, Dyken, Mark, & Abboud, 1993; Vgontzas et al., 1997). Furthermore, it has been shown that the high arousal present in depression is associated with increased beta activity, which adds further support to the hyper-arousal hypothesis of depression but further compromises sleep quality (Hall et al., 2000; Nofzinger et al., 2000). In a vicious cycle, increasing severity of depression, which is linked to greater dysregulation of arousal systems (Holsboer, 2000a, 2001) also exacerbates sleep disturbance (Vitiello et al., 2002) which in turn contributes to further dysregulation of arousal mechanisms and increased depressive symptomatology (Riemann et al., 2001). Therapies for depression aim to normalise the physiological imbalance in arousal and as a consequence, ameliorate the dysphoria and sleep disturbance which is prominent in MDD (Adrien, 2002).

Paradoxically, sleep deprivation is an effective remedy for depression as it has been found to exert a short term antidepressant effect, possibly by its ability to produce acute increases in serotonin (Adrien, 2002; Challet, Turek, Laute, & Van Reeth, 2001). Periodic use of sleep deprivation has also been shown to normalise sleep EEG with decreased sleep onset, suppression of REM sleep, increased restorative slow wave sleep, and a decrease in early morning waking (Clark, Frank, & Brown, 2001; Grozinger, Kogel, & Roschke, 2002; Hemmeter, Seifritz, Hatzinger, Muller, & Holsboer-Trachsler, 1995). These effects, however, are short lived as subjects revert

to their depressed state following their first night of recovery sleep. Chronic sleep deprivation, on the other hand, has the opposite effect and over time increases the risk of relapse and heightens the risk of suicide in depression (Dement & Vaughan, 1999; Holsboer-Trachsler & Seifritz, 2000).

Bright light therapy is a non-pharmacological intervention effective in ameliorating sleep and mood disturbance in conditions such as seasonal affective disorder, where circadian malfunction is prominent (Levitt, Lam, & Levitan, 2002). The ability of bright light to shift circadian timing with concomitant behavioural and emotional changes is well established (Armitage et al., 1999; Colwell, 1995; Middleton, Stone, & Arendt, 2002; Wallace, 1996; Wehr, Aeschback, & Duncan, 2001). For example, applying bright light in the early hours of the morning, close to the nadir of cortisol secretion, phase advances circadian rhythms, while bright light applied in the evening has a phase delaying effect (Boivin, Duffy, Kronauer, & Czeisler, 1994; Kubota et al., 2002). Early morning light therapy is the preferred treatment paradigm for use in depression as it lifts mood, which is typically low in the morning, possibly due to the suppression of melatonin (Badia, Myers, Boecker, & Culpepper, 1991; Terman et al., 1989). Given that cortisol has an arousing effect, the ability to delay nocturnal cortisol secretion using light therapy, may decrease early morning waking which is common in depression.

Traditional bright light therapy uses high intensities of light (approximately 10,000 Lux) but it has been found that even ordinary room light (approximately 180 Lux) can shift circadian rhythms (Boivin & Czeisler, 1998; Boivin, Duffy, Kronauer, & Czeisler, 1996). Of interest in this study, is the finding that very low intensity

(approximately 8 Lux) short wavelength light can also entrain circadian rhythms (Warman, Dijk, Warman, Arendt, & Skene, 2003). This research offers the potential that short wavelength low intensity light may be useful for shifting dysregulated cortisol rhythms in MDD. There is little evidence, however, that bright light therapy is effective in ameliorating sleep and mood disturbances in non-seasonal affective disorders despite the finding that cortisol, which is a marker of circadian rhythm, is dysregulated (Healy & Waterhouse, 1995; Wirz-Justice, 1995).

Antidepressant medications, as discussed previously, work partly via their ability to reinstate homeostatic regulation and normalise HPA over-activity (Barden et al., 1995; Bench et al., 1995). In addition to alleviating dysphoria, and exerting direct physiological effects on brain tissue, antidepressants have also been shown to normalise sleep irregularities (Armitage, 2000; Brunello et al., 2000; Mendlewicz, 1991). Congruent with the hyper-arousal hypothesis of depression, it is proposed that they exert their effects by decreasing nocturnal secretion of cortisol and stimulating the secretion of melatonin (Wallace, 1996). A common finding with effective treatment is the suppression of REM sleep and elongation of REM onset (Brunello et al., 2000; Murck et al., 2003; van Bemmel, 1997). Up to 40% of people, however, fail to respond to anti-depressant treatment or find the side effects intolerable (Montgomery, Baldwin, & Riley, 2002; Spigset & Martensson, 1999). Others, for various reasons, simply prefer to seek non-drug treatments for their depression and sleep problems.

While drug therapy may assist by normalising neurotransmitter secretion, and consequently improve sleep architecture, antidepressants are not effective in

reducing sleep onset, which is often delayed in depression because of increased arousal and excessive rumination (Morin, Rodrigue, & Ivers, 2003). Rather, cognitive therapies, which include relaxation methods, have been shown to be superior to drug therapies in their ability to address sleep initiation problems (Perlis, Smith, Cacialli, Nowakowski, & Orff, 2003). Up to 70-80% of people with sleep problems who use non-pharmacological interventions, such as biofeedback, relaxation training and cognitive and behavioural techniques, find them to be effective (Morin, Hauri et al., 1999).

### 3.10 The relaxation response and the treatment of mood and sleep disorders

Relaxation therapy is a proven non-pharmacological treatment for sleep and stress related disorders such as depression (Fennell, 1996; Morin, Hauri et al., 1999; Morin, Mimeault, & Gagne, 1999; Rosen, Lewin, Goldberg, & Woolfolk, 2000; Viens, De Koninck, Mercier, St-Onge, & Lorrain, 2003). Everly and Benson (1989) propose that stress responses can be reversed by repeated elicitation of the relaxation response, which, over time, can retrain a dysregulated arousal system and help to reinstate homeostasis. Relaxation responses have been shown to have calming effects on the autonomic nervous system, effectively reducing stress responses and increasing positive mood states (Leher, Carr, Sargunaraj, & Woolfolk, 1994).

According to Benson (1975) the relaxation response is a generalised phenomenon of reduced physiological arousal regardless of the technique used. Some studies, however, claim that different relaxation methods impact differentially on physiological parameters (Leher et al., 1994; Matsumoto & Smith, 2001), and elicit different feeling states (Matsumoto & Smith, 2001; Smith, Amutio, Anderson, &

Aria, 1996). For example, progressive muscle relaxation has been shown to have more somatic features (Leher et al., 1994) and to elicit feelings of 'disengagement' (Matsumoto & Smith, 2001), while deep breathing methods have been shown to increase mental awareness (Matsumoto & Smith, 2001; Smith et al., 1996), and meditation has been associated with 'prayerfulness', 'joy' and profound reductions in arousal (Smith et al., 1996; West, 1980).

Other studies find little to no differences between methods (Morse et al., 1977; Zeier, 1985) which supports Benson's (1975) claim that relaxation is a generalised reduction in physiological activity. According to Scheufele (2000), any changes attributable to various relaxation methods are 'superimposed' on this background of decreased physiological arousal. In addition, a common finding is that relaxation training, with repeated exposure to the relaxation response and frequent stimulation of the parasympathetic system, improves the strength of relaxation responses, dampens hyper-arousal mechanisms, and enhances clinical outcomes (Carney et al., 2000; Carson, Hathaway, Tuohey, & McKay, 1988; Cunningham, Brown, & Kaski, 2000; Lehrer et al., 2000; Lucini et al., 1997; Matsumoto & Smith, 2001; Orme-Johnson, 1973; Rosen et al., 2000; Zeier, 1985).

In direct contrast to stress responses, which reflect sympathetic and HPA activation, relaxation responses are induced by activation of parasympathetic pathways which produce reduced physiological arousal. Specifically, relaxation responses produce reductions in blood pressure (Agras, Taylor, Kraemer, Allen, & Schneider, 1980; Carson et al., 1988), heart rate, skin conductivity, and muscle activity and elicit mental quieting (Fried, 1990; Morse, 1993; Morse et al., 1977). Central cortical

changes include decreases in beta activity, especially in frontal areas, (Jacobs et al., 1996), and increases in alpha and theta activity (Arambula et al., 2001; Jacobs & Lubar, 1989; Schacter, 1977) which are evident during relaxation and persist into the 'awake' state in experienced meditators (Corby, Roth, Zarcone, & Kopell, 1978). Furthermore, positive mood states (Ford, Stoebel, Strong, & Szarek, 1982; Morse, 1993), and improvements in sleep quality (Agras et al., 1980; Rosen et al., 2000) are commonly reported by-products of relaxation training.

Krampen (1999) demonstrated that regular practice of autogenic relaxation could significantly enhance the effectiveness of psychotherapy in a group of clinically depressed psychiatric outpatients who were able to maintain their depression levels below clinical levels for up to 2 years post-treatment (Krampen, 1999). Lazar and colleagues (2000) used functional MRI to show that meditation increased cortical activity in prefrontal and limbic structures, which are areas of reduced activity in MDD. This supports the use of the relaxation response in the treatment of arousal disorders and suggests that relaxation therapy may help to restore neuronal damage caused by over stimulation of arousal systems.

Biofeedback training, which induces relaxation responses, has also been shown to directly impact on sleep quality. Using operant conditioning methods, Sterman (2000) showed that increasing 11-15Hz activity in the sensory motor strip in the awake state stimulated the production of sleep spindles during sleep, thereby improving sleep efficiency and stabilising sleep regulation. Hauri et al. (1982) also used biofeedback training in the alpha and theta ranges and improved sleep quality in a group of anxious insomniacs. In particular, they found that theta training, which is

associated with deep relaxation, was most effective for insomniacs with high anxiety levels as it helped to reduce night time wakings caused by high arousal. Reducing arousal, therefore, appeared to be the key to successful treatment (Hauri, Percy, Hellekson, Hartmann, & Russ, 1982). Offering further support for the use of relaxation therapies in the treatment of stress-related disorders, relaxation training has also been shown to produce permanent changes in autonomic functioning as evidenced by decreased cortisol secretion (McKinney, Antoni, Kumar, Tims, & McCabe, 1997) and attenuation of stress reactivity in response to stressful stimuli (Lucini et al., 1997; Orme-Johnson, 1973).

Relaxation therapies, particularly breathing techniques, have been shown to restore homeostatic balance by acting directly on the autonomic nervous system, further supporting their use in hyper-arousal disorders. Respiratory sinus arrhythmia, which is a measure of parasympathetic vagal tone, reflects the influence of breathing oscillations on heart rate variability (HRV) (Moser et al., 1994). Specifically, each inspiration stimulates the sympathetic branch of the vagus nerve and increases heart rate, while each expiration activates the parasympathetic branch with a concomitant decrease in heart rate (Lehrer et al., 2000; Vaschillo et al., 2002). High HRV reflects parasympathetic and sympathetic balance, indicating healthy regulation of the autonomic nervous system, which is predictive of adaptive coping in the face of stressors (Fabes & Eisenberg, 1997).

In stress related disorders, such as depression and anxiety, which are characterised by over stimulation of arousal systems, sympathetic inputs to the heart go unchecked as parasympathetic inputs are decreased. As a consequence, heart rate variability

decreases as rate increases (Moser et al., 1998), indicating a reduction in the ability to regulate autonomic control, which subsequently compromises adaptive processes and increases the risk of cardiac incidents in susceptible individuals (Gorman & Sloan, 2000). Relaxation therapy has been shown to enhance parasympathetic tone, therefore dampening sympathetic over-stimulation and as a consequence, re-establishing homeostasis (Sakakibara et al., 1994).

### 3.11 Photic stimulation in the treatment of mood and sleep disorders

Relaxation therapy has been shown to enhance both short and long term treatment outcomes in MDD (Krampen, 1999) and insomnia (Rosen et al., 2000) when used in conjunction with psychotherapy. Photic stimulation is a proven relaxation method with reliable anxiolytic effects which make it an appropriate choice for reducing arousal in stress related disorders such as MDD with accompanying sleep disturbance (Brauchli, 1993; Brauchli et al., 1995; Morse, 1993, 1994a; Ossebaard, 2000). Other studies provide direct evidence that photic stimulation is an effective treatment for MDD, decreasing dysphoria and enhancing cortical activity (Hammond, 2000; Kumano et al., 1996; Takigawa, 1988). As outlined in Chapter 2, Study 1 showed that audiovisual stimulation, like relaxation, effectively reduced symptoms of depression, anxiety, and psychological distress over time. Given previous research and the findings from Study 1, photic stimulation has the potential to be an effective adjunct in the treatment of psychiatric disorders such as mood and sleep disturbance, with some added advantages over traditional relaxation techniques.

Brainwave entrainment methods such as photic stimulation promise to be more effective than relaxation therapy in the treatment of mood disorders for a number of reasons. Firstly, their anxiolytic qualities can be used to address the arousal components of depression and help to re-establish homeostasis (Brauchli, 1993; Dieter & Weinstein, 1995; Morse, 1993; Ossebaard, 2000). Secondly, the phenomenon of photic driving can directly influence cortical neuronal activity and help to guide the brain towards normalacy (Jin et al., 1997; Joyce & Siever, 2000; Kikuchi et al., 2002; Kumano et al., 1997; Timmermann, Lubar, Rasey, & Frederick, 1999; Toman, 1941). Thirdly, unlike relaxation therapies, which take time and effort to master, increasingly popular mind-machines produce states of profound relaxation with little effort. Finally, photic stimulation has been shown, even at low levels of illumination, to reset circadian and diurnal rhythms, such as sleep-wake cycles, which are often dysregulated in major depression (Boivin & Czeisler, 1998; Middleton et al., 2002; Warman et al., 2003). Photic stimulation, therefore, may be effective in ameliorating not only mood disorders, but also the sleep disturbance that is commonly found in depression.

### 3.12 Summary

In this Chapter, depression has been conceptualised as a stress-related disorder, which allowed many of its symptoms to be understood as a function of dysregulated arousal systems that are disconnected from normal regulatory control mechanisms. It has also provided a rationale for the use of cost effective interventions such as photic stimulation and relaxation therapy. The ultimate aim of relaxation therapies for the effective treatment of hyper-arousal disorders is to re-train the autonomic

nervous system and re-establish sympathetic and parasympathetic balance which dampens HPA and LC/NE responses to stress.

In support of the use of photic stimulation for stress reduction, Study 1 found that audiovisual stimulation produced immediate relaxation responses but not long term effects to indicate enduring nervous system change. For example, immediate reductions in heart rate and skin conductance levels were evident during recording sessions, but remained essentially unchanged from baseline to follow-up. In Study 1, brainwave entrainment, relaxation, and sitting quietly with eyes closed all reduced arousal levels and produced immediate relaxation responses during recording sessions. Photic stimulation did show some small additional effects, with different frequencies of stimulation affecting mood and arousal in predicted directions. Alpha stimulation appeared to enhance positive affect after a number of sessions, while beta stimulation decreased positive affect and was accompanied by a small increase in skin conductivity, indicating and increase in physiological arousal. A major finding in Study 1 was that even in a non-clinical sample, depression, anxiety and psychological distress decreased over time, possibly due to the induction of relaxation responses in all groups, and contributed to non-specific effects such as the building of a therapeutic alliance and positive expectancies.

Study 1 showed that photic stimulation effectively reduced arousal. Photic stimulation, therefore, has the potential to be a useful tool in the treatment of mood and sleep disorders that are characterised by overstimulation of arousal systems. In addition, photic stimulation has all the necessary components proposed by Benson (1975) to elicit relaxation responses, plus the added contribution of brainwave

entrainment that can be used to shift abnormal brainwave states into more normal patterns of functioning. Given recent research on the ability of even low luminance light to entrain circadian rhythms, photic stimulation also offers the potential to directly impact on the regulation and timing of cortisol and melatonin secretion with further benefit to mood and sleep. Photic stimulation, therefore, may be more effective than simple relaxation techniques, like autogenic relaxation, in its ability to re-train over-stressed nervous systems, and help in the treatment of mood and sleep disorders.

In the next chapter, Study 2 will explore the capacity of photic stimulation to ameliorate dysphoria, and improve sleep, in a clinical sample of depressed individuals with sleep disturbance. It attempts to address some of the issues that may have interfered with the expression of relaxation and mood effects in Study 1 by providing participants with portable light devices that can be used at home on a regular basis. Consequently, Study 2 endeavours to maximise exposure to relaxation responses in order to facilitate training effects that will generalise beyond relaxation practice and beneficially impact on mood and sleep as arousal systems are normalised and homeostasis is re-established.

# CHAPTER 4

## Study 2: 'Photic stimulation and the treatment of mood and sleep disorders.'

### 4.1 Introduction

Depression has been conceptualised as a stress disorder (Chrousos & Gold, 1992) characterised by over-stimulation of the sympathetic noradrenergic system and dysregulation of the HPA system, with increased serum cortisol (Madjirova et al., 1995), mood swings, and often comorbid anxiety (Mineka et al., 1998; Wittchen et al., 2001; Yerevanian et al., 2001). These physiological disturbances also disrupt circadian behaviours such as sleep-wake cycles and eating, as normal regulatory processes are thrown into disarray (Armitage et al., 1999; Riemann et al., 2001; Wirz-Justice, 1995).

Recurrent episodes of depression exacerbate the problem as arousal systems become increasingly dysregulated and hyper-sensitive, and 'learn' to respond to threat with an exaggerated stress response (Everly & Benson, 1989; Post, 1992). Frequent elicitation of relaxation responses has been shown to be effective in the treatment of stress disorders, as it produces a physiological state which is incongruent with stress responses because it decreases arousal, and consequently helps to re-establish normal functioning (Benson, 1975, 1985; Everly & Benson, 1989). Photic stimulation promises to be an effective adjunctive therapy for use in mood and sleep disturbance because of its ability to induce relaxation responses. By the repeated induction of relaxation responses, photic stimulation can dampen an over-stimulated sympathetic system by enhancing parasympathetic activity, which, via reciprocal innervation, reduces HPA activity and over time, helps to reinstate homeostasis.

## 4.2 Review of Study 1's findings

Previous research has demonstrated clear entrainment responses to photic stimulation (Silberstein, 1995b; Toman, 1941) with beneficial effects on mood (Brauchli et al., 1995; Glicksohn, 1986; Morse, 1993, 1994a; Ossebaard, 2000) (see Chapter 1). Study 1 attempted to demonstrate the differential effects of various frequencies of stimulation on mood and cortical activity, but only small to moderate non-significant effects were found. As discussed in Chapter 2, entrainment responses were present in many participants receiving audiovisual stimulation (AVS), with demonstrated increases in EEG amplitude, not only at their frequency of stimulation, but also in the other bandwidths of interest. In Study 1, frequency of stimulation influenced mood to a small degree. Alpha stimulation increased positive affect, while beta stimulation decreased positive affect over experimental trials. Detection of these small effects, however, was hampered by low statistical power. The main finding of Study 1, which informs the present study, is that photic stimulation was equally as effective as autogenic relaxation in its ability to induce relaxation responses, and to reduce symptoms of depression and anxiety, over the course of the study, even in a non-clinical sample. These findings indicate that photic stimulation may be an effective adjunctive tool in the treatment of stress-related disorders, like depression, because of its ability to reduce arousal, and ameliorate symptoms of depression and anxiety.

In summary, the findings of Study 1 revealed that 13Hz stimulation tended to increase positive affect and subjective feelings of relaxation, while 22Hz AVS tended to decrease positive affect and increase physiological arousal. Furthermore, photic stimulation was found to be an effective relaxation therapy as 5Hz, 13Hz and 22Hz AVS produced relaxation effects similar to those induced by autogenic

relaxation. In particular, 5Hz AVS appeared to induce the greatest reduction in depression and anxiety symptoms indicating that it may have been more effective at eliciting relaxation responses than the other frequencies. The small to moderate treatment effects observed in Study 1 may have been due to the use of a sample with little pathology and, consequently, little room for change within mood and arousal parameters. In the present study larger effects sizes are anticipated as a clinical sample, who are clearly characterized by dysregulated mood, sleep and arousal, but who have more opportunity for improvement, will be used.

Similar to Study 1, Study 2 also attempts to demonstrate frequency following effects to determine if the phenomenon of brainwave entrainment contributes to relaxation and mood effects. Previous research has shown that brainwave frequencies are positively correlated with arousal and reflect different affective states. Therefore, it is hypothesised that by artificially inducing certain brainwave frequencies using brainwave entrainment techniques, concomitant arousal and mood effects will be induced (see Chapter 1 for a more complete review). For example, theta (5-7Hz) is associated with drowsiness (Niedermeyer, 1993; Strijkstra et al., 2003) and found during deep relaxation (Arambula et al., 2001; Dierks, Maurer, & Zacher, 1989; Jacobs & Lubar, 1989). Alpha rhythms (8-13Hz) also indicate cortical deactivation which differs from theta in that they do not accompany drowsiness, but rather a state of rest and expectant alertness (Basar et al., 1997; Pfurtscheller et al., 1996). Alpha is also associated with positive mood states (Brown, 1970; Nowlis & Kamiya, 1970). Beta rhythms (≥14Hz) reflect cortical desynchronisation which indicates cortical activity and is present during task engagement (Micheloyannis et al., 2002; Ray & Cole, 1985; Wrobel, 2000). They are also positively related to arousal (Bonnet &

Arand, 2001) and anxiety (Wendy. Heller et al., 1997; Pizzagalli et al., 2002), and if present during the night, elevated beta compromises sleep efficiency (Nofzinger et al., 2000). In order to maximise these mood effects and achieve training effects, participants will be exposed to photic stimulation frequencies on a regular basis over an extended period, which will also address some of the limitations of previous research.

### 4.3 Limitations of Study 1

A criticism of earlier research assessing the effects of audiovisual stimulation on mood and relaxation was the use of short exposure times to entraining stimuli (Brauchli et al., 1995). To address this problem some studies used repeated exposures of short duration (von Gizycki et al., 1998). Other studies more thoroughly assessed the therapeutic utility of audiovisual entrainment by using longer exposure times over a number of sessions. These studies all attest to the ability of audiovisual stimulation to induce immediate relaxation effects (Morse, 1993, 1994a; Morse et al., 1977), produce reductions in state anxiety (Ossebaard, 2000), and improve academic performance (Budzynski, Jordy, Budzynski, Tang, & Claypoole, 1999; Carter & Russell, 1993; Joyce & Siever, 2000; Patrick, 1996), but failed to demonstrate long term changes needed to validate the use of photic stimulation in the clinical arena.

In clinical studies compliance is often a problem (Michalak, Hayes, Wilkinson, Hood, & Dowrick, 2002). Researchers rely on participants being motivated to attend the AVS sessions, or else they use a 'captive' sample such as a school setting wherein participants are easily accessible. Most relaxation techniques require effort,

motivation, and an investment of time in order for them to be effective since repeated exposure to relaxation responses is necessary to retrain arousal systems to respond in a less reactive manner. In clinical settings, relaxation skills are taught and clients are then encouraged to practice the skill at home on a daily basis, in order to facilitate training effects that generalise beyond the relaxation practice itself so that adjustments in sympathetic reactivity can be realised. Aware of the issues of compliance and motivation, other studies have supplied participants with light devices which could be used at home on a regular basis over an extended period of time in order to maximise relaxation and therapeutic effects (Anderson et al., 1997; Noton, 1997).

Generalisation of relaxation responses beyond relaxation practice may have been hampered in Study 1 by requiring participants to visit the university for their relaxation sessions. While every attempt was made to maximise exposure to relaxation by asking participants to attend sessions up to 3-4 times per week, some could not do this. While most participants demonstrated immediate relaxation responses during relaxation and AVS, there was little evidence to suggest that the physiological quieting attained during sessions endured over time. Resting heart rate, skin conductance and EEG showed within-session relaxation responses, but changed little from session to session. To address this problem, Study 2 provided participants with take home light masks in order to encourage regular use and maximise exposure to relaxation responses, in order to facilitate training effects which would retrain over-stimulated arousal systems and achieve enduring and beneficial changes to mood and sleep.

## 4.4 Aims of Study 2

The main aim of this study was to assess the effectiveness of photic stimulation as an adjunctive therapy in the treatment of major depression with associated sleep disturbance. Specifically, the study set out to determine if there were differential effects of particular photic stimulation frequencies on mood and sleep. The main question asked in the study was a clinical one; "Does exposure to particular photic stimulation frequencies for up to twenty minutes a day over a period of one month have beneficial effects on mood and sleep and if so, what frequencies of stimulation are most effective?" By providing clients with their own light mask to use at home it was anticipated that relaxation responses would be maximised and the hypothesised effects on mood and sleep would become evident.

**4.4.1 Electroencephalography**: Another aim of the current study was to determine if evidence could be found for the commonly reported phenomenon of frontal alpha asymmetry in depression. Davidson and colleagues have provided a large body of research showing that depressed subjects display hemispheric asymmetry in the EEG with increased alpha in the left midfrontal cortex relative to the right midfrontal cortex, indicating deactivation of the left frontal cortex (Davidson, 1992, 1993, 1998b; Davidson & Henriques, 2000; Davidson et al., 2002; Tomarken & Keener, 1998). Non-depressed subjects fail to show this asymmetry or display the opposite pattern, with increased right midfrontal alpha in comparison to left midfrontal alpha, signifying increased activation of the left frontal cortex (Henriques & Davidson, 1991). These findings are amply supported by brain imaging (Drevets, 2003; Mayberg, 2003; Soares & Mann, 1997) and neuropsychological studies (Austin,

1995; Austin et al., 1999; Shenal et al., 2003) which show deficits in frontal and limbic brain regions. Thus depression is accompanied by a decrease in functioning of the left pre-frontal cortex. In the current study, therefore, which used a clinically depressed sample, it was anticipated that participants would display an increase in left midfrontal alpha (F3) relative to right midfrontal alpha (F4) at baseline, and that alpha asymmetry would be positively correlated with depression level and be unrelated to group membership.

In addition, while Henriques and Davidson (1991) found evidence of alpha asymmetry in depressed subjects with higher alpha power in left midfrontal regions in comparison to the control group, they did not find any difference between depressives and controls in absolute alpha power in right midfrontal regions. Therefore, it was predicted in the current study that left midfrontal alpha amplitude (8-13Hz, at F3) would be higher in comparison to the normative database, while right midfrontal alpha amplitude (8-13Hz, at F4), would be similar to the normative database.

Successful treatment of depression is often accompanied by normalisation of cortical anomalies (Drevets, 1998; George, Ketter, & Post, 1993). In fact, placebo induced remission shows changes in prefrontal areas similar to those found with active therapy, with increases in frontal metabolism (Mayberg et al., 2002). Davidson, however, found that while cortical asymmetries decrease with remission of depression, the pattern of asymmetry persisted even when improvements in mood occurred. Thus according to Davidson, the pattern of frontal cerebral asymmetry is a trait like marker for depression (Henriques & Davidson, 1990). In the current study,

therefore, it was anticipated that cerebral asymmetry would be relatively stable over time and independent of treatment outcome.

**4.4.2 Brainwave entrainment:** Similarly to Study 1, the current study also aimed to demonstrate entrainment effects with increases in EEG amplitude at the frequencies of stimulation. In Study 1 it was hoped that evidence of entrainment would be found in order to support the presence of a physiological response to AVS. Unfortunately, there was very little statistical evidence of frequency following in response to AVS, even during stimulus presentation, with only small to moderate trends observed.

To explore this further, EEG amplitude in occipital sites (Mean P3-O1 and P4- O2) was averaged across the 12 experimental sessions in Study 1 for each AVS group (5Hz, 13Hz, and 22Hz) and visually compared to baseline measures (see Figures 4.1, 4.2, and 4.3). In the 4-6Hz bandwidth (Figure 4.1) it was observed that the beta group was the only group to demonstrate an increase in 4-6Hz, not the theta group as predicted, while the alpha, relaxation and control groups showed an overall decline in 4-6Hz during AVS in comparison to baseline. In the 12-14Hz bandwidth (Figure 4.2), all active AVS groups showed a small increase across the twelve experimental sessions in comparison to baseline and follow-up, while the relaxation and control groups showed the opposite pattern with a decrease in 12-14Hz across the twelve sessions. For the 21-23Hz bandwidth (Figure 4.3), only the beta group showed an increase in 21- 23Hz EEG amplitude in comparison to baseline, suggesting possible entrainment effects were present at higher beta frequencies and not at lower frequencies. While these observed effects were small to moderate, and non-

significant, some interesting patterns did emerge. The beta group consistently showed increases in EEG amplitude across the three bandwidths tested, suggesting that higher entrainment frequencies affected the EEG in a global way, possibly inducing greater cortical relaxation responses as evidenced by increased theta and alpha frequencies, and possibly even entrainment effects, as suggested by increased amplitude in the 21-22Hz bandwidth for the beta group only. In order to address the issue of low power in Study 1, the current study increased experimental power by increasing subject numbers in order to more effectively test for evidence of entrainment effects.

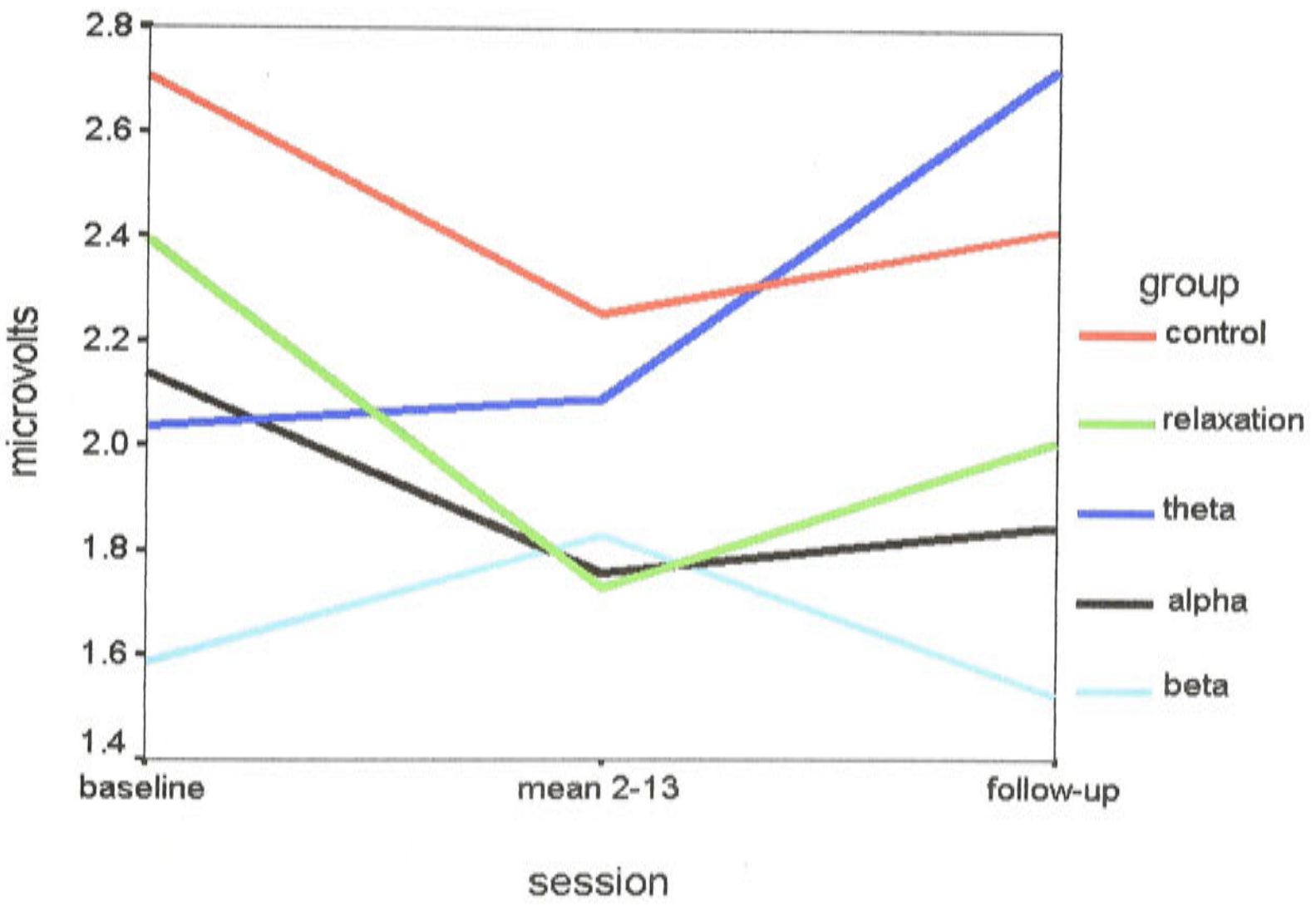



Figure 4.1 Comparison of mean amplitude in occipital 4-6Hz at baseline, during photic stimulation (sessions 2 to 13), and at follow-up (n=30).

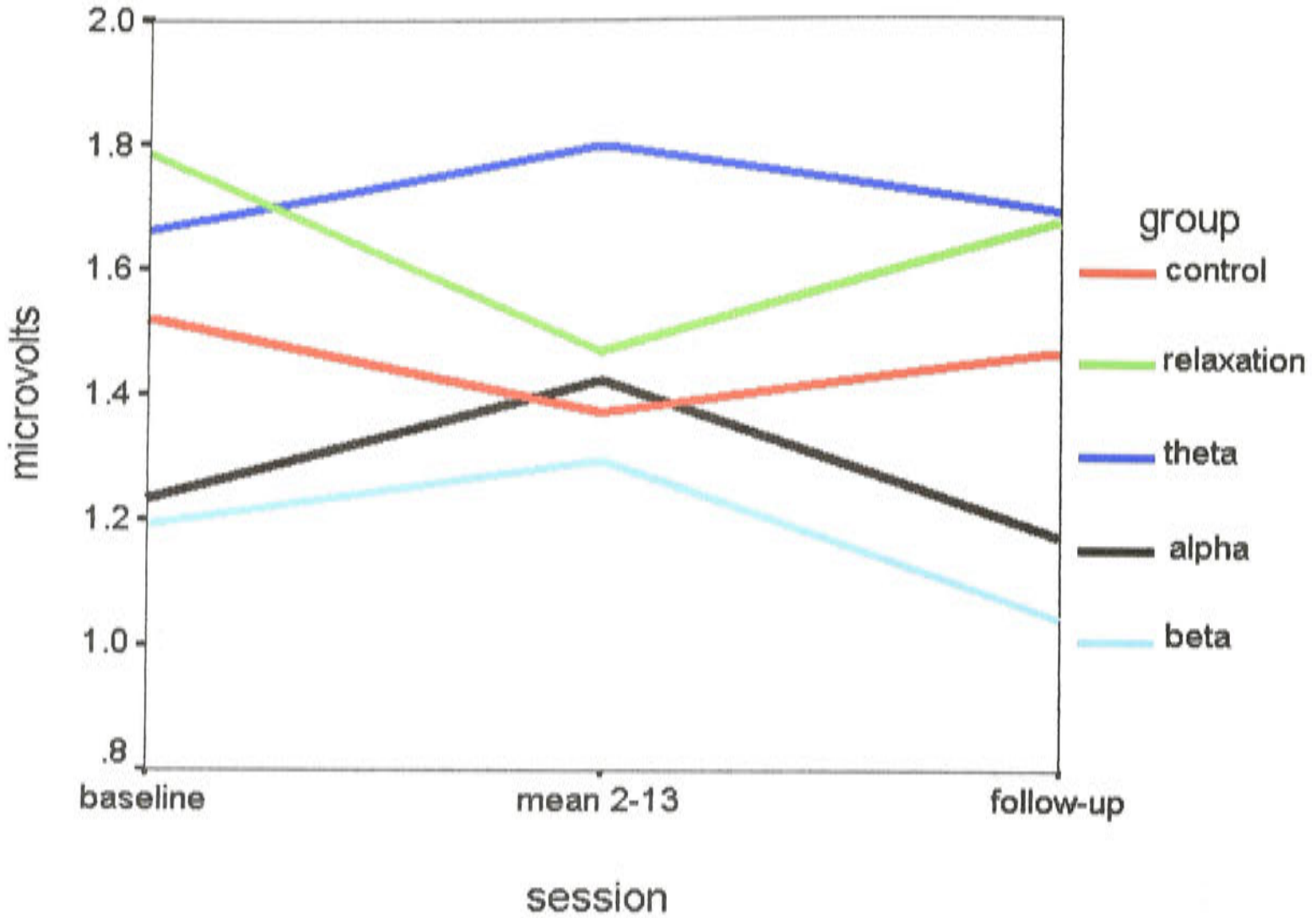



Figure 4.2 Comparing mean amplitude in occipital 12-14Hz at baseline, during photic stimulation (sessions 2 to 13), and at follow-up (n=30).

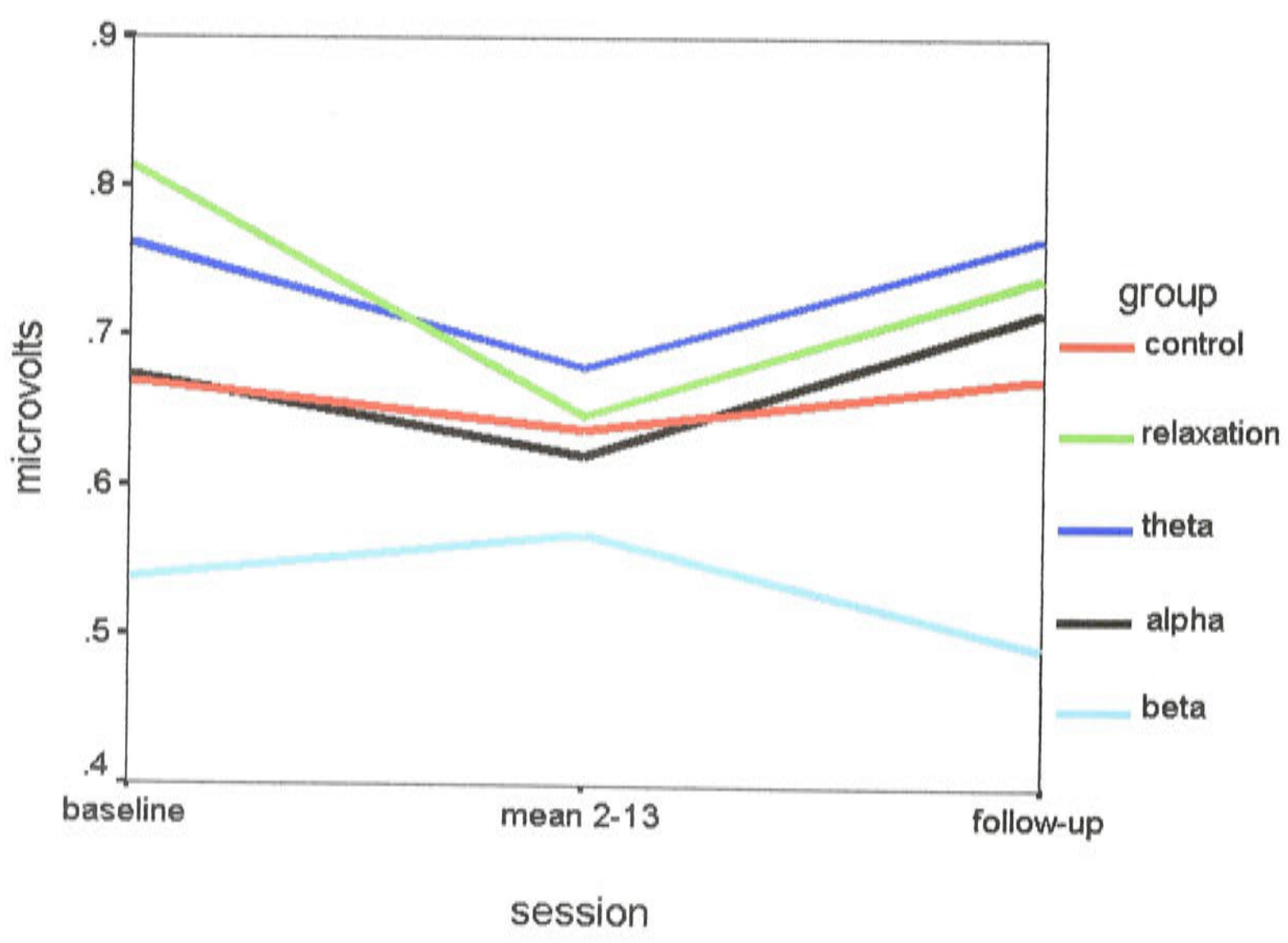



Figure 4.3 Comparing mean amplitude in occipital 21-23Hz at baseline, during photic stimulation (sessions 2 to 13), and at follow-up (n=30).

As discussed previously, the lack of evidence for frequency following responses in Study 1 may have been due to the frequencies of stimulation used. Frequency following is more likely to occur with stimulation frequencies in the 8-12Hz bandwidth, which are close to the person's peak alpha frequency. Therefore, the second study used a procedure suggested by Pockberger (1985), and participants were exposed to a 10Hz photic stimulation for 3 minutes while their EEG was simultaneously recorded in order to assess frequency following responses across the cortex.

Photic driving responses are most likely to occur in the occipital cortex, but both auditory and photic driving responses have been shown to recruit to distant cortical areas (Glicksohn, 1986; Kikuchi et al., 2002; Morse, 1993; Mundy-Castle, 1953; Neher, 1961). If photic driving responses are recruited from occipital to frontal brain regions and subsequently change oscillatory neuronal behaviour in these areas in the direction of the driving frequency, then it may be possible to use photic stimulation in the treatment of frontal lobe disorders such as depression, which are characterised by frontal lobe deactivation. Therefore, in the current study, it was hypothesised that photic driving responses would be demonstrated with increases in 10Hz EEG amplitude not only in occipital cortical regions, but also in frontal regions as the response is recruited across the cortex. In addition, Pockberger showed that entrainment responses are related to depression severity, with attenuation of the response with recovery. Thus it was expected that frequency following responses would be positively related to level of depression.

The small to moderate non-significant entrainment effects present in Study 1 were observed at both entrainment frequencies and in distal bandwidths. Therefore, some people demonstrated first order and harmonic driving responses at frequencies distant from their dominant frequency. For example, those receiving 22Hz AVS demonstrated driving responses across all bandwidths of interest. Given this, in the current study it would be advantageous to assess entrainment responses to the experimental photic stimulation frequencies used in addition to the 10Hz stimulation outlined above. However, in order to maintain the double blind condition this was not feasible, as the experimenter conducting the EEG's needed to remain naïve to the participants' allocated frequencies. There was some concern that the blind would be exposed if participants were allowed to use their Lightmask during an EEG session as often the frequency of stimulation is apparent in occipital EEG when driving responses are being observed.

Study 1 showed that AVS successfully elicited relaxation responses, possibly due to the relaxation features present coupled with entrainment effects. Photic stimulation fulfils Benson's requirements for inducing relaxation as it helps to distract the mind by providing a repetitive stimulus on which to focus. It also attenuates external sensory input by wearing headphones and glasses. In addition, entrainment effects help to guide the brain into a relaxed state. As a consequence, muscle tension decreases as relaxation responses ensue. In the current study, with enhanced power, and regular daily exposure to entrainment stimuli, it was anticipated that brainwave entrainment effects would be maximised. If frequency following contributes to treatment outcomes, then it was anticipated that regular daily exposure to a specific stimulus may, over time, produce demonstrable changes in the EEG as training

effects become evident. Thus if entrainment effects are present, it was hypothesised that those receiving 5Hz, 13Hz and 22Hz photic stimulation would show increases in their eyes closed EEG at 5Hz, 13Hz and 22Hz, respectively.

**4.4.3 Subjective relaxation:** Cortical EEG frequencies generally reflect arousal levels. Lower theta frequencies are associated with deactivation and drowsiness, alpha rhythms with relaxed alertness, and beta frequencies signal an actively engaged and aroused mind (Niedermeyer, 1993). In Study 1 there was a general increase in subjective relaxation over sessions for all groups after AVS, with a trend towards those receiving 13Hz (alpha) AVS to report feeling more relaxed. This trend was in expected directions as alpha rhythms are often increased during relaxation and usually accompany positive mood states (Basar et al., 1997; Brown, 1970; Morse et al., 1977).

In the current study, however, subjective relaxation ratings were not immediately contingent with photic stimulation use. Rather, subjective relaxation ratings were obtained when participants attended the hospital for their EEG recordings. It was anticipated that regular daily exposure to the different photic stimulation frequencies would impact differentially on relaxation responses, with greater relaxation anticipated for those receiving theta and alpha stimulation, and lower relaxation effects for those receiving beta or continuous light. Therefore, if the different experimental frequencies used in Study 2 produce enduring entrainment effects and subsequent relaxation responses, then it was anticipated that those receiving photic stimulation at 5 and 13Hz would report higher subjective relaxation during the experimental phase than those receiving 22Hz photic stimulation, continuous light or

no therapy. Consequently, it was expected that subjective relaxation would correlate positively with theta and alpha bandwidths and negatively with beta EEG. The immediate effects of photic stimulation were assessed in the current study by asking participants to rate their subjective well-being after each light therapy session, as discussed below.

**4.4.4 Mood** (PANAS): In the first study, there was an overall decrease in positive affect across sessions. There were some trends in predicted directions, however, with the alpha group showing a sustained increase in positive affect after session four and the beta group showing consistently low positive affect across sessions, but these effects did not reach significance. Also, in Study 1, certain items on the PANAS were endorsed more than others, with participants endorsing items such as 'interested', 'enthusiastic', 'inspired', 'proud', 'alert', 'active' and 'determined' on the positive affect scale, and 'distressed', 'upset', 'irritable' and 'nervous', on the negative affect scale, independent of group membership.

Previous research shows that relaxation enhances well-being (Benson, 1975; Ford et al., 1982; Marshall & Bentler, 1976; Morse, 1993; Morse et al., 1977; Smith et al., 1996; Stoyva, 1989). In addition, alpha rhythms, which commonly accompany relaxation responses, are generally associated with feelings of well-being and positive affect (Glicksohn, 1986; Hinrichs & Machleidt, 1992; Plotkin, 1979; Rosenfeld, Baehr, Baehr, Gotlib, & Ranganath, 1996; Rosenfeld et al., 1997; Saxby & Penniston, 1995; Shimokochi, 1996; Slatter, 1960; Wackermann, Pütz, Büchi, Strauch, & Lehmann, 2002). Given that photic stimulation reliably produces relaxation responses, it was hypothesised that in the current study, photic stimulation

would enhance positive affect and decrease negative affect during experimental trials. Specifically, it was predicted that those in the active photic stimulation groups, 5Hz, 13Hz, and 22Hz, would report higher levels of positive affect and lower levels of negative affect than those in wait list (WL) or continuous light (CL) groups.

As previously stated, in Study 1 those receiving 13Hz (alpha) AVS reported higher positive affect after session 4, while those receiving 22Hz (beta) AVS consistently reported lower positive affect across sessions, accompanied by greater skin conductivity. It appears therefore that alpha stimulation is more pleasurable and less arousing than beta stimulation. Therefore, in the current study it was hypothesised that those receiving 13Hz photic stimulation would report higher levels of subjective well-being immediately after light therapy than those receiving 22Hz photic stimulation.

**4.4.5 Depression:** Major depression is often a recurrent disorder characterised by periods of relapse and remission (Maj et al., 1992). Many people recover from an episode of major depression over time, in the absence of therapeutic intervention (American Psychiatric Association, 2000; Ross, Quitkin, & Klein, 2002; Solomon et al., 1997). In Study 1, which used a non-clinical sample, depression levels decreased for all groups, except the alpha group, over the course of the study. As previously discussed, the alpha group started the study with depression scores below the norm and consequently were affected by floor effects. The decrease in depression symptoms had little to do with the particular AVS frequencies participants were receiving. Rather, it may have been due to the relaxation responses elicited by AVS or simply to changes in mood and depression levels over time.

Congruent with the 'hyper-arousal' hypothesis for depression, any intervention that reduces arousal by eliciting relaxation responses, has the potential to be beneficial in the treatment of arousal disorders such as depression and anxiety. Non-specific therapeutic effects and expectancy effects, however, usually play a role in any clinical study and contribute to treatment effects (Bench, 2001; Frank, 1982a; Jensen & Karoly, 1991). In addition, as acknowledged above, depression changes over time and improvement may occur regardless of the intervention received. Therefore, in the current study it was anticipated that depression would decrease for all groups, including the control and wait list groups, over the study period because of the variable nature of depression over time and the influence of therapeutic and expectancy effects. In Study 1, it was shown that AVS, regardless of frequency, elicited relaxation responses. In Study 2, therefore, it was hypothesised that depression would decrease more for those in the active photic stimulation groups, regardless of the specific frequency of stimulation, than for those in the control or continuous light groups.

**4.4.6 Anxiety:** Anxiety is often comorbid with depression. In Study 1 anxiety also decreased from baseline to follow-up in all groups, except the alpha group. Again, the alpha group were affected by floor effects and therefore did not show any change in anxiety over time. While greater reductions in anxiety were seen in the theta, beta and relaxation groups than in the control group, the observed effects were small and not significant; but still suggest that active therapy is more effective than no intervention.

Therefore, in the current study, using a clinical sample, anxiety was also expected to decrease over sessions, firstly because of the relaxing effects of photic stimulation and secondly as a function of different photic stimulation frequencies. It was hypothesised that photic stimulation at 5Hz would be most effective for participants with moderate to high anxiety as it would assist in reducing excess cortical beta which often accompanies states of hyperarousal and anxiety (Chapotot et al., 1998; Matousek, 1991). Photic stimulation at 22Hz was hypothesised to be effective for participants with low anxiety, as it would help to ameliorate frontal lobe deactivation, which often accompanies depression, and help to raise cortical activity and thus, increase motivation and energy levels.

**4.4.7 Symptom Severity**: Symptom severity (global severity index- Symptom Checklist 90-R) decreased in all groups in Study 1. The global severity index provides an intensity score which reflects the impact of symptoms from all nine subscales of the SCL-90-R. Therefore, it is a useful clinical indicator of the impact of symptoms regardless of the source. Because of the ability of AVS to elicit relaxation responses and consequently reduce symptom severity, it was anticipated that global severity index would decrease more for the active photic stimulation groups than the WL or CL groups.

**4.4.8 Sleep and photic stimulation:** As discussed in Chapter 3, depression is often accompanied by sleep disturbance (Brunello et al., 2000; Papadimitriou, Dikeos, Daskalopoulou, & Soldatos, 2002; Thase, Simons, & Reynolds, 1996). In this study, depression is viewed as a hyperarousal disorder with accompanying physiological dysregulation. Techniques which reduce arousal and elicit relaxation responses,

therefore, have the potential to be beneficial for mood and sleep disturbance. Study 2, therefore, also investigated the utility of photic stimulation to ameliorate sleep problems which are often exacerbated by high levels of arousal (Nofzinger et al., 2000).

Relaxation techniques are frequently used in the treatment of sleep disorder (Dement & Vaughan, 1999; Shapiro, Bootzin, Figueredo, Lopez, & Schwartz, 2003; Viens et al., 2003). Given that AVS was found to be an effective relaxation tool in the last study, regardless of frequency of stimulation, it was hypothesised that photic stimulation at 5Hz, 13Hz and 22Hz would be more effective at reducing sleep onset latencies and night wakings and improving sleep efficiency than continuous light (CL) or no therapy (WL). Consequently, those in the 5Hz, 13Hz and 22Hz groups are expected to rate their waking mood higher than those in the WL, or CL groups. In addition, if photic driving responses at various frequencies of stimulation were to exert differential effects on sleep in this current study, then it was expected that 5Hz photic stimulation would be most effective for those people who report difficulty falling asleep, as it would assist in decreasing higher cortical activity and help in the production of theta frequencies which are required for sleep onset.

Furthermore, given Sterman's (1981, 2000) work on operant conditioning of the 'sensory motor rhythm' (SMR 12-15Hz) and its demonstrated ability to improve sleep and decrease night wakings, it was expected that photic stimulation at 13Hz would be most effective for treating night wakings and therefore improving sleep efficiency over the course of the study. Finally, it was hypothesised that photic stimulation at 22Hz would have less impact on the amelioration of sleep onset latency in

comparison to the 5Hz and 13Hz group due to its tendency to increase beta activity, which is at odds with sleep onset, but would continue to have more impact than WL or CL because of induced relaxation effects.

## 4.5 Method

**4.5.1 Participants:** The sample consisted of sixty participants drawn from the Canberra community. Participation was voluntary with participants responding to advertisements in the local newspaper or, alternatively, being referred by health care workers who had received information about the study requesting suitable referrals. The sample included 22 males and 38 females with an age range of 18 to 82 years, (M=41.70, SD=14.30). There was an attrition rate of 8% (n=5). Two participants moved interstate, one participant left the study after commencement of anti-depressant medication, the eldest participant, who was in the wait-list group, died suddenly from pneumonia, and one participant withdrew because of increased work commitments. Two participants withdrew from the waitlist group, two from the continuous light group, and one from the 22Hz group. In general, the active light therapy groups retained participants suggesting that photic stimulation at least sustained people's interest. No participant reported leaving the study because of an inability to tolerate the light therapy.

Handedness was assessed because of previous convention set by Davidson and colleagues when investigating cerebral asymmetry and emotion (Davidson et al., 1990; Davidson & Fox, 1989; Henriques & Davidson, 1990, 1991; Tomarken, Davidson, & Henriques, 1990). Fifty two participants were right handed, 5

participants were left handed and the remaining 3 participants were ambidextrous as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971).

**4.5.2 Selection criteria**: Participants were admitted to the study if they met the DSM-IV criteria for mild to moderate major depression with symptoms of sleep disturbance (see Appendix E for Selection Criteria). In order to recruit participants with unipolar depression only, respondents were excluded from the study if they had a history of bipolar disorder or psychosis. People taking anti-depressant or anxiolytic medication were also excluded because of the confounding effects these medications can have on the EEG (Knott, Mahoney, Kennedy, & Evans, 2002). Because photic stimulation has been known to induce epileptic seizures in some light sensitive epileptics, people with a history of epilepsy were not permitted to enter the study.

While there are studies which report the usefulness of high frequency photic stimulation in the treatment of migraine (Anderson, 1989), random allocation of participants to groups prohibited control of the particular photic stimulation frequencies participants would receive, therefore, people with a history of migraine were excluded from the study. Furthermore, people who reported snoring 'more nights than not' were also excluded from the study because of the high likelihood they could be suffering from sleep apnoea (Bardwell et al., 2000). Traditionally, relaxation therapies have little impact on the amelioration of sleep apnoea (Dement & Vaughan, 1999). Approximately 80 respondents were excluded because of failure to meet admission criteria.

Beck Depression Inventory II (BDI-II) scores ranged from 2 to 46 (M=23.48, SD=11.04) on admission to the study. Despite intake criteria, 18% of participants commenced the study with BDI-II scores in the minimal range (0-13), 22% fell within the mild range (14-19), 33% fell within the moderate range (20-28), and 27% were in the severe range of depression (29-63). Most participants, 72%, reported a depression history of 12 months or longer, (M=102 months, SD=121).

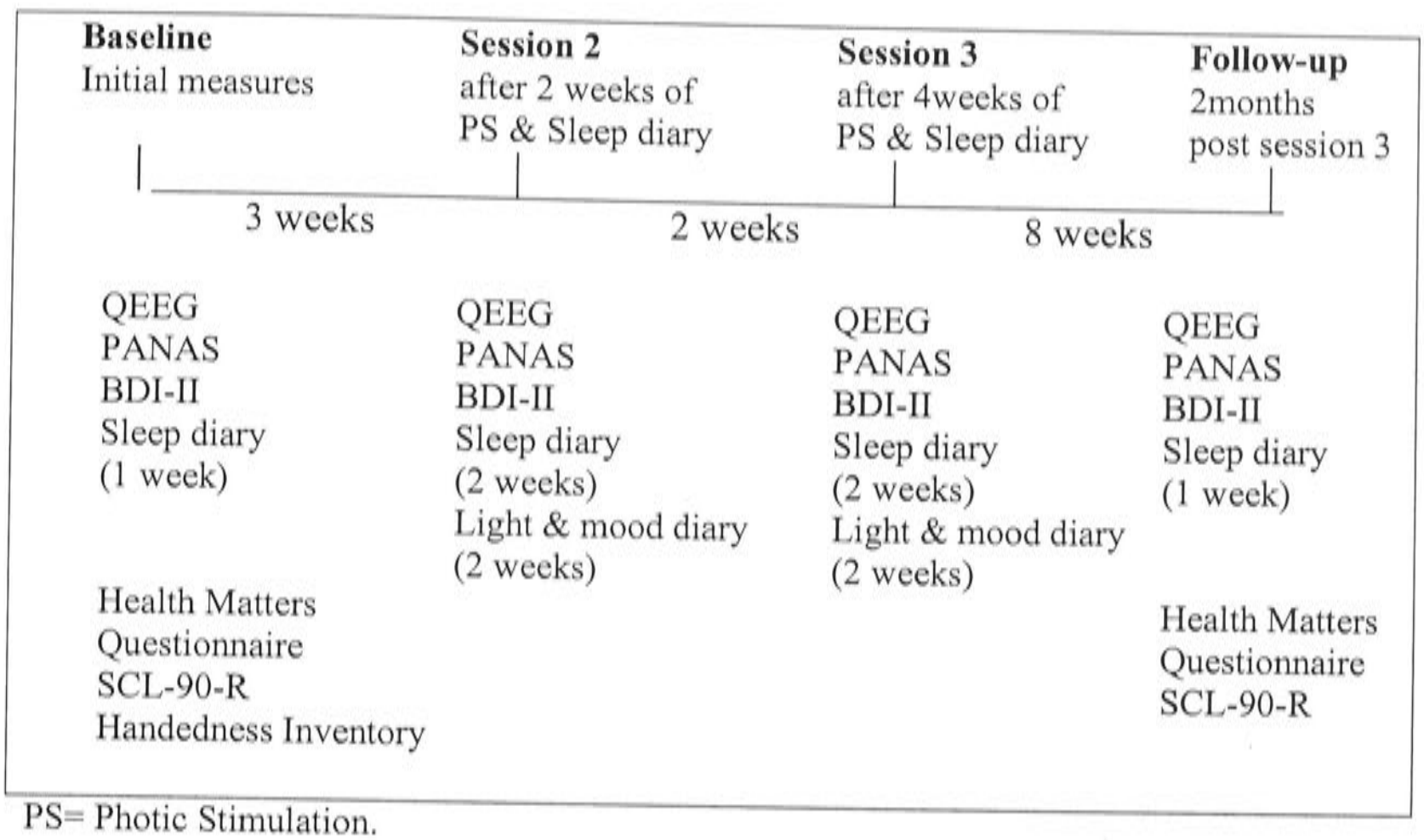
Symptom Checklist 90-R (SCL-90-R) anxiety T-scores ranged from 37 to 81 (M=64, SD=10.85) on admission. Sixty three percent of participants had SCL-90-R anxiety scores greater the 1SD above the norm. Despite intake criteria, on initial assessment 1 participant reported no sleep problems.

At initial assessment, only 18% of participants reported having been hospitalised in the last 12 months for minor surgery, cancer treatment, or treatment for cardio-pulmonary disease. One participant had been hospitalised for a suicide attempt. A substantial proportion of participants, 33%, reported living with a chronic illness that included ailments such as hypertension, Systemic Lupus Erythematosus, asthma, genital herpes, arthritis, breast cancer, or glaucoma. One participant was a Vietnam Veteran with a long history of Post Traumatic Stress Disorder who reported being stable at the time of entry into the study. In addition, 45% of participants reported having been affected by a major life event in the last 3 months, reporting events such as relationship break-ups, death or illness of friend or family member, moving house, changing jobs or financial stressors.

Twenty percent of the sample were smokers, but only 12% of these reporting smoking 10 or more cigarettes per day. Only a small proportion, 13% (n=8) reported using recreational drugs, with four clients claiming they used marijuana on a regular basis ranging from everyday to a few times per week. Some participants disclosed they used ecstasy, heroin or cocaine on occasion. No one in the study reported using heroin, cocaine or ecstasy on a regular basis. These participants were asked to abstain from recreational drug use for 24 hours prior to EEG recordings, while all participants were asked to refrain from caffeine intake for 4 hours prior to EEG recordings to minimise EEG attenuation associated with caffeine intake (Gilbert, Dibb, Plath, & Hiyane, 2000).

## 4.6 Design

A double blind, placebo controlled, 5 by 4 repeated measures mixed design was used. Participants were randomly allocated to one of five groups; a wait list control group (WL); a continuous light group (CL), who were exposed to photic stimulation of 64Hz which is the highest setting on the Lightmask™ and is beyond the flicker fusion frequency, or one of three active photic stimulation groups, receiving photic stimulation at either a 5Hz, 13Hz, or 22Hz. As can be seen in Figure 4.4, measures of EEG, mood, and depression, were collected four times over a 3 month period; at baseline, twice during the month of experimental trials, i.e. 2 weeks after using the lights and/or keeping a sleep diary, and at the end of the 4 weeks, and finally at follow-up 2 months after the completion of light therapy.




PS= Photic Stimulation.

Figure 4.4 Experimental design for study 2 showing measures taken at each session. During the 4 week intervention phase (includes session 2 & 3) participants in the light groups kept sleep diaries and a light and mood diary. Participants in the wait-list group kept a sleep diary only.

## 4.7 Measures

On initial interview demographic information, measures of health behaviours, life events, mood, and sleep hygiene were presented together in the 'Health Matters Questionnaire' (see Appendix F). At all sessions, baseline, session 2, session 3 and follow-up, measures of mood, subjective relaxation, depression and EEG were obtained. Psychological status was also assessed using the Symptom Checklist 90-R at baseline and follow-up only. In addition, participants kept sleep diaries, recording sleep hygiene variables, as discussed below, plus light and mood diaries, recording time of Lightmask use, and mood before and after Lightmask use, for up to six weeks during the study.

**4.7.1 Subjective relaxation:** Subjective relaxation was assessed at each measurement session prior to EEG recording. Participants were asked to indicate on a 10 point Likert scale, 1(feeling very relaxed and calm) to 10 (feeling very anxious and uptight), how relaxed and calm or anxious and uptight they felt 'right now' (see Appendix F, 'Health Matters Questionnaire', p. 4). In order to obtain a measure which was reflective of the descriptor 'subjective relaxation', this variable was reverse scored so that high scores corresponded with high levels of relaxation and low scores with low scores of relaxation.

**4.7.2 Mood (PANAS).** Mood was assessed at every session using the Positive and Negative Affect Schedule (PANAS) developed by Watson and Clark (1988) (see Appendix F, 'Health Matters Questionnaire', p. 4). This is a 20 item scale with 10 items assessing positive affect and 10 items assessing negative affect. Respondents were asked to indicate, on 5 point Likert scale (1 = very slightly or not at all, to 5 = extremely) "to what extent you have felt this way in the last week".

**4.7.3 Depression:** Depression level was assessed at every session using the Beck Depression Inventory II (BDI-II) (Beck, Steer, & Brown, 1996). The BDI-II is a 21 item self-report instrument used to assess depression severity. It assesses emotional, cognitive, somatic and behavioural components of depression. Respondents are required to indicate, on a 4 point Likert scale (0-3) which statement in each group best describes them and they way they have been feeling 'during the past two weeks, including today'. Summed scores provide a depression severity score within the following ranges, 0-13 minimal, 14-19 mild, 20-28 moderate, and 29-63 severe depression.

The BDI-II boasts high internal consistency reporting alpha coefficients of .92 (Beck et al., 1996). This was confirmed in the present study with an alpha reliability of .89 to .92 across administrations. Test re-test reliability is also reported to be high, at .93 which makes it a good instrument to use in a repeated measures design (Beck et al., 1996). An added advantage of the BDI-II is that it has been designed to be consistent with the DSM-IV diagnostic criteria for major depression.

**4.7.4 Psychological status:** Psychological status was assessed on two occasions, at baseline and at follow-up, using the Symptom Checklist 90-R (SCL-90-R). As outlined in Chapter 2, the SCL-90-R is a 90 item inventory which has been extensively used in both 'normal' and clinical populations. Respondents were asked to indicate on a 5 point Likert scale the extent to which they had been distressed or bothered by each of the 90 listed problems over the last 7days. The SCL-90-R reports good reliability and validity with alpha coefficients for internal consistency reported for the depression scale at 0.90 and for the anxiety scale, 0.88, and high test-retest reliability, 0.82 (Derogatis & Savitz, 1999). The subscales used in this study were anxiety, and the global severity index, and are measured as T-scores with mean of 50 and SD of 10. The depression subscale was used to validate the use of the Beck Depression Inventory with Pearson's correlation coefficients between the two scales of $r = .64$, $p < .01$, at baseline, and $r = .35$, $p < .01$, at follow-up. According to these moderate correlations the SCL-90-R and the BDI-II do not assess the same symptoms of depression. Nevertheless, the selection of a clinical sample was confirmed with 85% of participants with SCL-90-R depression T-scores of 60 or higher at baseline.

**4.7.5 Sleep hygiene:** Sleep hygiene was assessed using a retrospective self-report method (see Appendix G). All participants were required to keep sleep diaries for a period of six weeks; at baseline for one week after the initial assessment and prior to commencing light therapy; during the 4 weeks of experimental trials; and finally, for one week prior to follow-up measures. Respondents were asked to fill in their sleep diary in the morning on waking and record, the time of 'lights out' the previous night, estimation of time to fall asleep (sleep onset latency), estimated 'number of wakings' during the night and an estimation of the amount of 'time spent awake' during these times, final waking time in the morning, reasons for waking if remembered, and any general comments relating to the last nights sleep. From these measures a sleep efficiency measure was calculated (ratio of total time asleep to total time in bed, expressed as a percentage). Clients were also asked to rate, on a 10 point Likert scale, 1 (the worst I have ever felt) to 10 (the best I have ever felt), how they felt on waking.

**4.7.6 Light therapy and mood diary**: In addition to keeping sleep diaries, those in the light conditions kept a record of the time of day they used their light mask and how they felt before and after light mask use, in order to assess if time of day of light mask use affected mood or sleep parameters, and also to enable a check on the consistency of light mask use (see Appendix H). In addition, to assess if particular photic stimulation frequencies had an immediate effect on subjective well-being participants were asked to indicate on a 10 point Likert scale, 1 (the worst I have ever felt) to 10 (the best I have ever felt), how they felt before and after light mask use.

**4.7.7 Menstrual cycle:** Because of the unusual finding in Study 1 where positive affect was found to be positively correlated with week of menstrual cycle, week of menstrual cycle was again measured at baseline in an attempt to replicate this finding. As outlined in Chapter 2, women were asked to indicate the current week (1-4) of their menstrual cycle, with week one corresponding to the onset of menses, subsequent weeks 2 and 3, and week 4 indicating the final week of the cycle or encompassing the remaining time until the beginning of the next cycle. Correlations were performed only on women who indicated a menstrual cycle week, n=23 (see Appendix F, Health Matters Questionnaire, p. 5).

## 4.8 Apparatus

**4.8.1 Photic stimulation**: During EEG recording photic stimulation at 10Hz was delivered using a portable light and sound machine (XCELR8R II) manufactured by 'Mind-gear'™. The device has 4 light diodes for each eye; white, green, red and blue, arranged in a circular array that delivers a low intensity (approximately 10 Lux) synchronised pulsed (on/off) light stimulus through closed eyelids.

**4.8.2 Lightmask**: Participants in the light conditions were each given a commercially available 'Lightmask'™ and a recharging unit to take home and use (see Appendix I for description). Each light mask was preprogrammed at the designated frequency for each group by an independent person so the experimenter would remain blind to group allocation. The Lightmask™ is a fully programmable easy to use portable photic stimulation device that delivers a synchronised pulsed (on/off) light stimulus of a set frequency via two red diodes (one for each eye),

through closed eyelids. Light intensity was set low to medium (approximately 10-15 Lux) in order to find a level which could be tolerated by most people.

In order to control for possible variation in photic driving responses in the EEG due to time of day effects, participants were asked to use their light mask in the evening, and preferably, just prior to retiring and optimally, at the same time each day. On average, participants tended to use their light mask between 9 and 10pm. In order to maintain the double blind condition, as discussed above, EEG was not assessed during 5Hz, 13Hz or 22Hz photic stimulation with the Light mask.

**4.8.3 Electroencephalography:** EEG was data was recorded using a 16 channel 'Minset'™ data acquisition device and stored on hard disk for further analysis. The Mindset used in this study was tested and calibrated by the manufacture prior to study commencement. Calibration was checked using a 50μV signal during and also at study completion. Some signal attenuation was found at 4Hz and below with approximately 10-20% signal attenuation in the 3-4Hz bandwidth, however, there was accurate signal detection for the remaining1Hz bins (5-22Hz) used in the study.

Data was sampled at 256Hz, with band pass filters set between 3 and 30Hz. An Electro-Cap™ was fitted according to instructions using 'Electro-Cap Gel'™ and an abrasive agent, 'Nu-Prep'™, in order to reduce impedance levels to 5Kohms or below. On occasion this was not possible, and impedance levels were kept below 10Kohms prior to EEG recording (Andreassi, 1995; Kaiser, 2000; Niedermeyer & Lopes Da Silva, 1993). Referential EEG measures were recorded from sixteen sites (Fp1, Fp2, F3, F4, F7, F8, C3, C4, T3, T4, T5, T6, P3, P4, 01, and 02) across the scalp in

accordance with the International 10/20 System (Homan, Herman, & Purdy, 1987; Jasper, 1958) using a linked ear reference montage (Davidson, 1988; Kaiser, 2000). Participants were seated comfortably in an upright chair approximately 1 metre from the Mindset and out of view of the video monitor display.

Raw EEG data were processed using SKIL™ software version 2.05 (Sterman & Kaiser, 2000). SKIL™ provides an automated artifact removal algorithm that removes variation from Fp1 caused by eye blink and movement. In addition, all raw EEG files were visually inspected to detect and remove movement artifacts present in other leads. Digitised EEG data were then subject to Fast Fourier Transform and decomposed into individual 1Hz frequency bins. Data was then exported for further statistical analysis. To ensure consistency in manual artifact removal a subset of raw EEG files were assessed and inspected for artifacts by an independent psychologist working in the EEG field. Pearson's correlation coefficient was used to compare time lengths of EEG files after artifact removal. Using this method inter-rater reliability was .87.

SKIL™ also provides a normative database. This database is comprised of repeated EEG measures from 135 people ranging in age from 18-55years, 80% percent male and 20% female. Subjects were recruited from the wider community and included students, laboratory personnel, and U.S Air Force personnel. Handedness was assessed using the Edinburgh Handedness Inventory (Oldfield, 1971), and subjects were screened for medical history, recent life events, and drug use. Time of day corrections are provided for use with the eyes closed and eyes open conditions. The

normative database also allows for comparison with eyes closed, eyes open, and two active task conditions such as reading and arithmetic.

## 4.9 Procedure

**4.9.1 Recruitment:** Subjects were recruited over a 12 month period from July 2001 to July 2002. Participants were either self-referred, responding to advertisements placed in a local newspaper or to information in pamphlets left at doctors' and psychologists' offices, or directly referred by health practitioners. Preliminary screening was conducted over the telephone to ascertain eligibility for entry into the study. Using a tick list of depression and sleep disorder symptoms, participants were admitted to the study if they met minimal requirements for mild to moderate major depression and also reported some symptoms of sleep disturbance as defined by DSM-IV (see Appendix E for Selection Criteria).

All sessions were conducted in a clinical room in the Psychology Department at The Canberra Hospital. During the initial interview, participants were told that the study was investigating the effects of photic stimulation or light flicker on mood and sleep disturbance. In order to keep expectancy and placebo effects constant, participants were informed that photic stimulation had been reported to have beneficial effects on mood and sleep but that there was little controlled research regarding its clinical usefulness, and that the present study was attempting to address this issue.

Participants were informed that it was a double blind study, and they would not know what frequency of photic stimulation they were receiving, nor the intended effects of the stimulation until the end of their participation, nor would the

experimenter recording their EEG know the particular light frequency they were receiving. Participants were shown the Lightmask™ that would be used in the study. Next they were informed that some frequencies would be very high and some very low and that the perception of 'light flicker' was not always possible; but nevertheless, photic stimulation had been shown to affect brainwave functioning. Research regarding the most beneficial frequencies for the treatment of mood disorders and the frequencies used in the current study were not discussed. Random allocation to groups and the waitlist condition were explained. Experimental procedures were outlined in detail prior to written consent being obtained.

Next, participants completed the 'Health Matters Questionnaire', the Beck Depression Inventory-II (Beck et al., 1996), the Edinburgh Handedness Inventory (Oldfield, 1971), and the Symptom Checklist 90-R (Derogatis, 1977).

Following this, EEG measures were obtained as previously outlined. Because of 'time of day' effects on the EEG, sessions were scheduled as much as possible at the same time each day (Cummings et al., 2000; Lafrance & Dumont, 2000; Lafrance et al., 2002; Sterman, 1998). Before baseline recording commenced, participants were asked to sit quietly for a number of minutes with their eyes closed in an attempt to induce a habituation response and minimise artifact. When participants indicated they were feeling quite relaxed a series of three minute recordings were done; eyes closed, eyes open, and 10Hz photic stimulation. Prior to the eyes closed and photic stimulation recordings, participants were instructed to sit quietly with their eyes closed and to try to minimise the amount of eye movement. Prior to the eyes open condition participants were instructed to fix a 'comfortable gaze' on the picture in

front of them. In an attempt to maintain a relaxed gaze without undue strain on the eyes, participants were told they could blink if necessary, but to avoid unnecessary eye movement or blinking. A small A4 size nature picture (a rainforest scene, a lake scene or a snow scene) was placed approximately 2 metres in front of the client slightly below eye level to help fix the gaze and avoid unnecessary eye movement. Up to 30 seconds was allowed after the initiation of a new task before recording commenced to allow for state changes and to minimise artifact in the EEG. If artifact contamination appeared excessive, the initial eyes closed condition was repeated at the end of recording, thus allowing for comparison with the first eyes closed and ensuring reliable baseline measures had been obtained.

Eyes open EEG was recorded in the absence of specific hypotheses in order to assess the normal functioning of the EEG. It is usual to observe attenuation of occipital alpha on eyes open and with task engagement (Hammond, 2002; Niedermeyer & Lopes Da Silva, 1993; Sterman, 1996). It also enabled comparison of the eyes open condition with the normative database provided with the SKIL™ software which facilitated meaningful feedback to clients at the end of the study.

Finally, participants were shown how to fill in their baseline sleep diary, and random allocation to group was done. If participants were in a light therapy group, an appointment for collection of their lightmask was made for the following week. In order to maintain the double blind condition the lightmasks were programmed by a colleague privy to the allocated photic stimulation frequencies. At the end of this process participants were given the opportunity to ask any further questions. All participants received an information pack to take away containing information about

the study, the researchers, sleep diaries, light and mood diaries, and diary keeping instructions (see Appendix J, for 'Participant Information Sheet').

During sessions 2 and 3, participants completed the PANAS and gave a subjective relaxation rating prior to EEG measures. At follow-up, all measures were re-done, and a final EEG obtained. Participants were debriefed by the second experimenter regarding the particular photic stimulation frequency they had received and the intended effects on mood and sleep. At the end of the study, participants were given material to take away, which included information about managing depression, guidelines for good sleep, and a list of references for obtaining information about photic stimulation and treatment of mood and sleep disturbance.

## 4.10 Results

Assumptions of linearity, normal distribution and homogeneous variance, were checked graphically by means of residual plots. Data was also screened for outliers using visual inspection of box plots and Mahalanobis's distance. Data analysis was done using the SPSS V.10 statistical package and repeated-measures multivariate analyses of variance (MANOVA) was used to test hypotheses using a .05 alpha level. Effect sizes are reported using partial eta squared ($\eta^2$). Means and standard deviations for variables used in hypotheses testing are located in Appendix K.

**4.10.1 Electroencephalography:** Processed EEG data was assessed for alpha attenuation in the occipital cortex on eyes open to ensure a 'normal' desynchronisation response was present. All participants demonstrated alpha attenuation to varying degrees. Untransformed mean magnitude ($\mu$V/Hz) EEG data

tended to be positively skewed. Natural log transformation remedied this and is the recommended transform for mean EEG (μV/Hz) data (Kaiser, 2000). Mean log EEG magnitude (μV/Hz) was used in all EEG analysis except alpha asymmetry which used a power measure. Broadband EEG variables, as recommended by Kaiser (2000), were used in some statistical analysis; theta (mean 5-7Hz), alpha (mean 8-13Hz) and beta (mean 14-20Hz). Because of increased EEG amplitude from frontal to posterior sites, correlational analyses used frontal (mean Fp1, Fp2, F3, F4, F7 & F8), midline (mean T3, T4, C3 & C4), or posterior (mean P3, P4, T5, T6, O1, & O2) broadband measures, or global theta, alpha or beta measures (means of all 16 sites).

Prior to using EEG data in hypotheses testing, mean log EEG magnitude for eyes closed was assessed for expected patterns of response using a four way MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and session (1-4), site (Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2), and band (theta 5-7Hz, alpha 8-13Hz, and beta 14-20Hz) as the within groups variables. There was a main effect for site, $F(9,38) = 42.42$, $p < .05$, $\eta^2 = .90$, revealing an increase in EEG amplitude moving from the front to the back of the cortex which is in accordance with expected patterns. There was also a main effect for band, $F(2,45) = 148$, $p < .05$, $\eta^2 = .86$, with the greatest amount of amplitude in the alpha band, followed by the theta band and finally, the beta band, as expected. In addition, there was a significant site by band interaction, $F(18,29) = 85.50$, $p < .05$, $\eta^2 = .98$, with the largest amplitudes occurring in the alpha band at the posterior sites, showing the well known phenomenon of occipital alpha peak frequency (Berger, 1976 reprint of 1934; Kaiser, 2000; Niedermeyer & Lopes Da Silva, 1993). There was no main effect for group.

**4.10.2 EEG alpha asymmetry:** Alpha asymmetry was assessed using the broadband alpha EEG variable, 8Hz to 13Hz, transformed into log power ($\mu V^2/Hz$), in accordance with that used by Henriques & Davidson (1991). Based on previous work by Davidson and colleagues, it was hypothesised that left midfrontal alpha would be greater than right midfrontal alpha in this depressed sample. This hypothesis was tested using a three way MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and site (F3 & F4) and session (1 to 4) as the within groups variables.

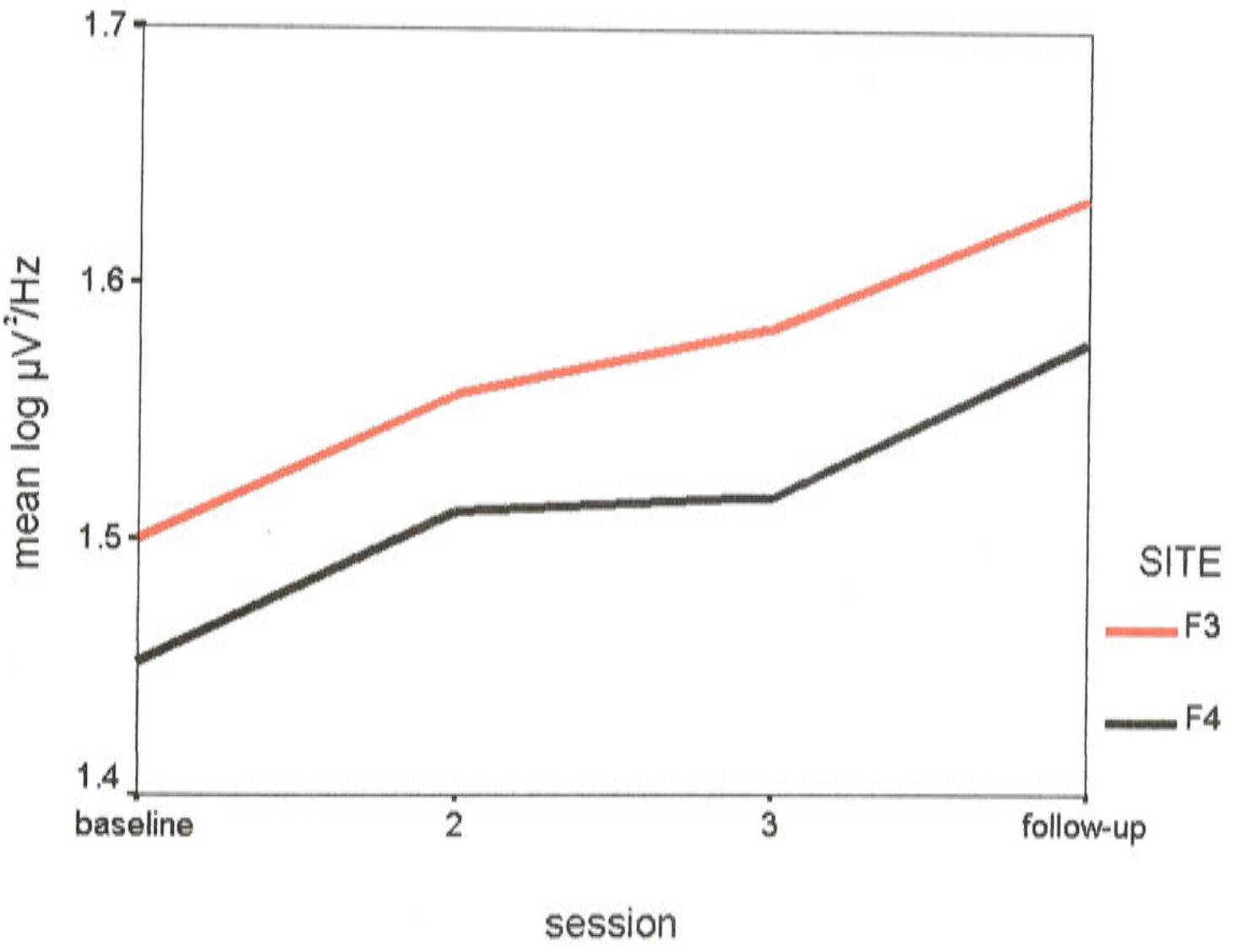



Figure 4.5 Mean log alpha power ($\mu V^2/Hz$) at F3 and F4 across sessions, showing higher left midfrontal alpha in comparison to right midfrontal alpha (n=51).

As can be seen in Figure 4.5 a main effect for site was revealed with mean log alpha power at F3 significantly higher than mean log alpha power at F4, $F(1,46) = 16.34$, $p < .05$, $\eta^2 = .26$. While alpha power increased across sessions this was not significant suggesting that alpha asymmetry was relatively stable across time, $F(3,44) = 2.31$, $p > .05$, $\eta^2 = .14$. Group membership did not influence alpha asymmetry as predicted, $F(4,46) = 1.1$, $p > .05$, $\eta^2 = .09$, and there were no interaction effects. Thus this sample of depressed clients showed left midfrontal deactivation relative to right midfrontal activation which persisted across sessions, indicating a trait like character as proposed by Davidson.

**4.10.3 Comparison to Normative Database:** Comparison of alpha (8-13Hz) EEG magnitude (μV/Hz) to the SKIL normative database showed that participants did not have higher left midfrontal alpha in comparison to the norm, with a mean standard deviation for the sample of -1.40 (SD= .57) at F3 accompanied by low right midfrontal alpha (F4) with a mean standard deviation of -1.43 (SD= .56). Overall the sample was situated within 2SD of the normative database in the alpha bandwidth across all 16 scalp sites. Further exploration found mean EEG magnitude to be lower than the normative database in all frequency bands across the scalp with the most prominent finding that of low frontal theta.

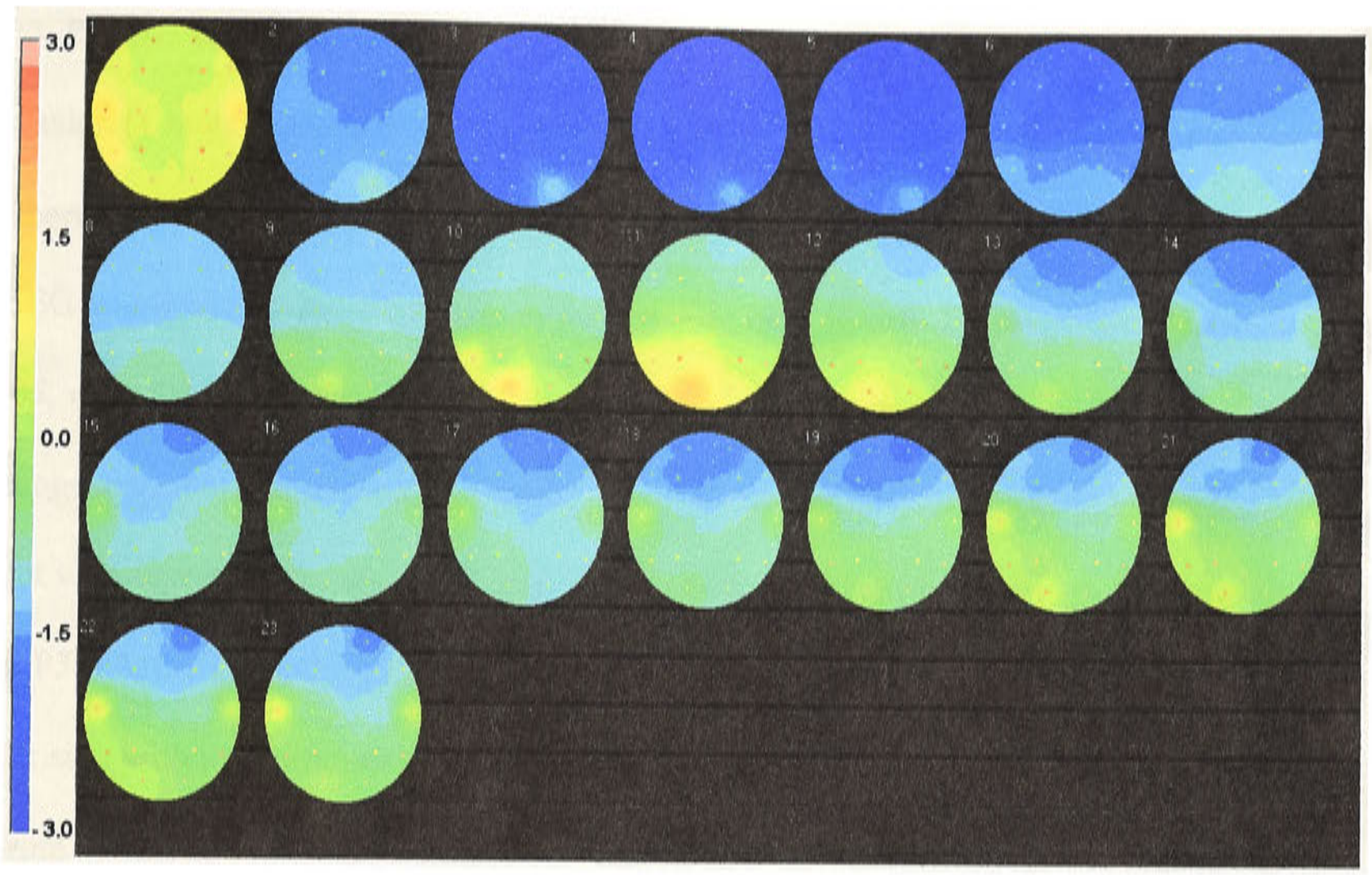



Figure 4.6 Topographical brain map eyes closed EEG in 1Hz frequency bins showing low frontal theta (5-7Hz) in comparison to the normative database (standard deviations) in a 56year old female.

Figure 4.6, is a topographical brain map of 1Hz frequency bins showing the typical pattern of low frontal theta across 5-7Hz found in this sample. Approximately 45-50% of participants demonstrated low frontal theta at Fp1, Fp2, F3 and F4 of less than 2SD or more below the norm. The sample also displayed low frontal beta, with approximately 30-35% of participants showing beta magnitudes of 2 or more standard deviations below the norm at Fp1, Fp2, F3, and F4.

**4.10.4 Brainwave entrainment:** It was anticipated that participants would demonstrate photic driving in response to a 10Hz photic stimulus, not only in occipital regions, but also in frontal regions with recruitment of the response across the cortex. This was tested using a four way MANOVA, with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and site (Fp1, Fp2, F3, F4, C3, C4,

P3, P4, O1, and O2), condition (eyes closed vs. 10Hz photic stimulation), and session (1 to 4) as the within groups variables, using mean log 10Hz EEG (μV/Hz). There was a main effect for condition with photic stimulation demonstrating higher EEG amplitude across all sites in comparison to eyes closed, $F(1,46) = 45.26$, $p < .05$, $\eta^2 = .50$. This effect was uncomplicated by session or group membership suggesting that it was somewhat stable across time and unaffected by light therapy, but was dependent on site, with larger driving responses noted in the occipital cortex at P3, P4, O1 and O2, $F(9,38) = 2.66$, $p < .05$, $\eta^2 = .39$. There was also a main effect for site, with a significant linear effect showing increased EEG amplitude moving from frontal to occipital sites as previously noted, $F(9,38) = 69.34$, $p < .05$, $\eta^2 = .94$. There was no main effect for group, $F(4,46) = 1.94$, $p > .05$, $\eta^2 = .14$.

Photic stimulation responses were varied with some participants showing no entrainment while others showed marked entrainment. Figure 4.7a shows baseline eyes closed EEG in a 22 year old female participant with a BDI-II score of 41. Her response to 10Hz photic stimulation is clearly visible in Figure 4.7b with maximal response evident in posterior leads. Many participants also showed clear harmonic responses to 10Hz photic stimulation. Figure 4.8a shows a spectral plot of eyes closed baseline EEG for the same 22year old female with 11Hz dominant alpha frequency clearly visible at O1, O2, P3 and P4. Her response to 10Hz photic stimulation is very evident in Figure 4.8b with increased amplitude in the 10Hz frequency bandwidth across the cortex, and clear 20Hz harmonic responses visible, not only in occipital leads, but recruiting into frontal leads.

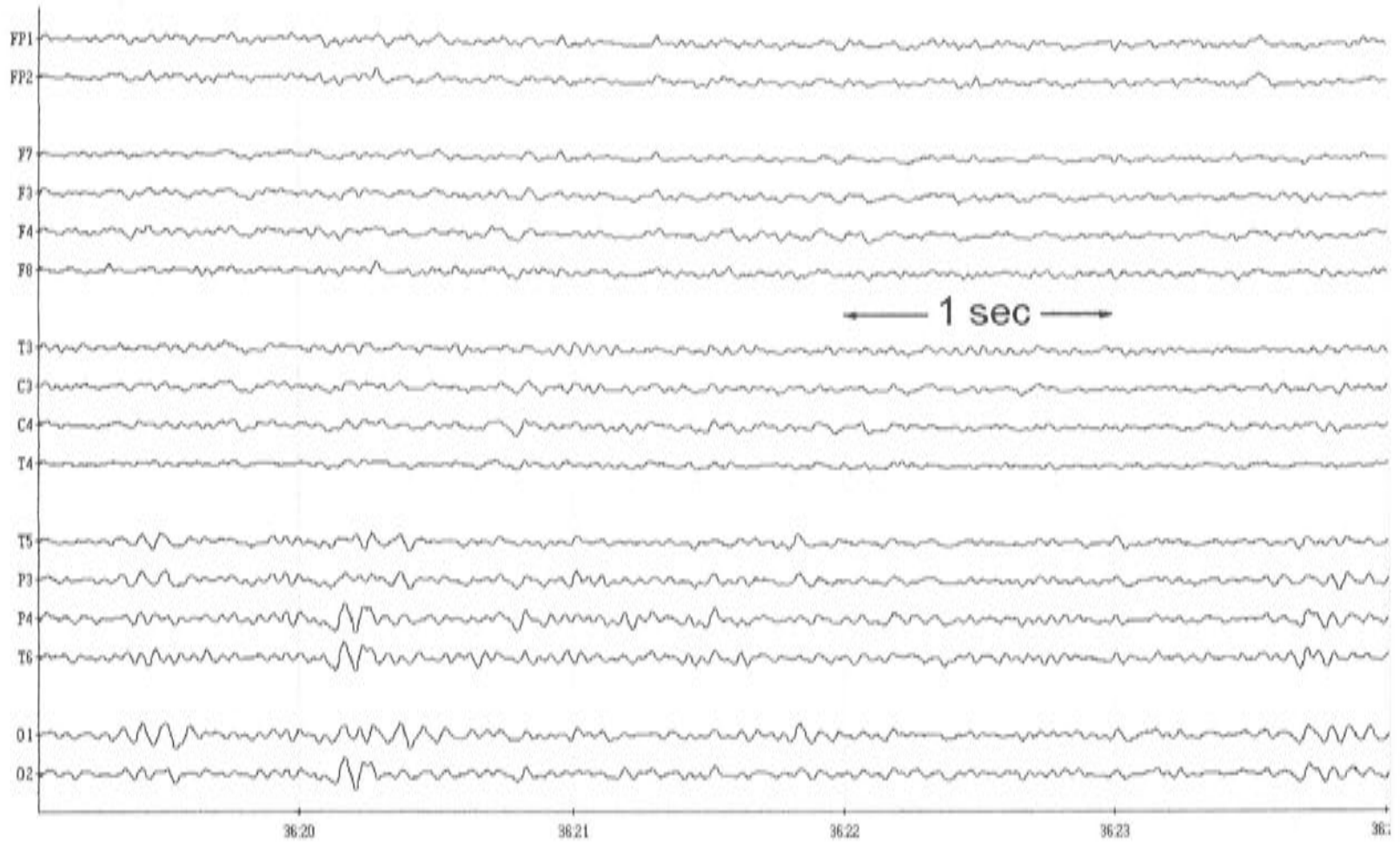



Figure 4.7a Eyes closed EEG across 16 scalp sites (y axis) in a 22 year old, right handed, depressed female, BDI-II=41.

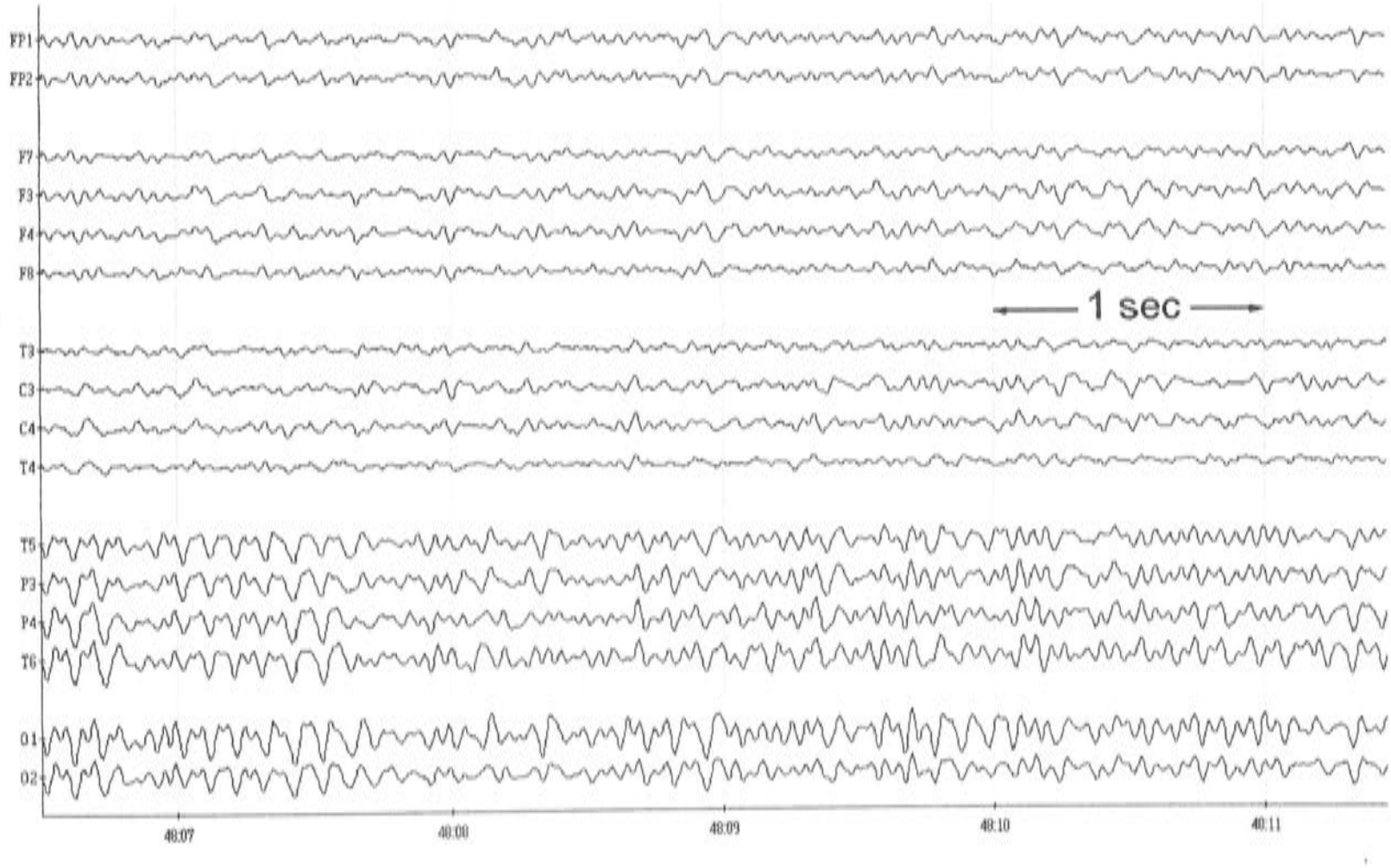



Figure 4.7b Eyes closed EEG showing 10Hz photic stimulation response particularly in occipital leads, and across the cortex in the same 22 year old female.

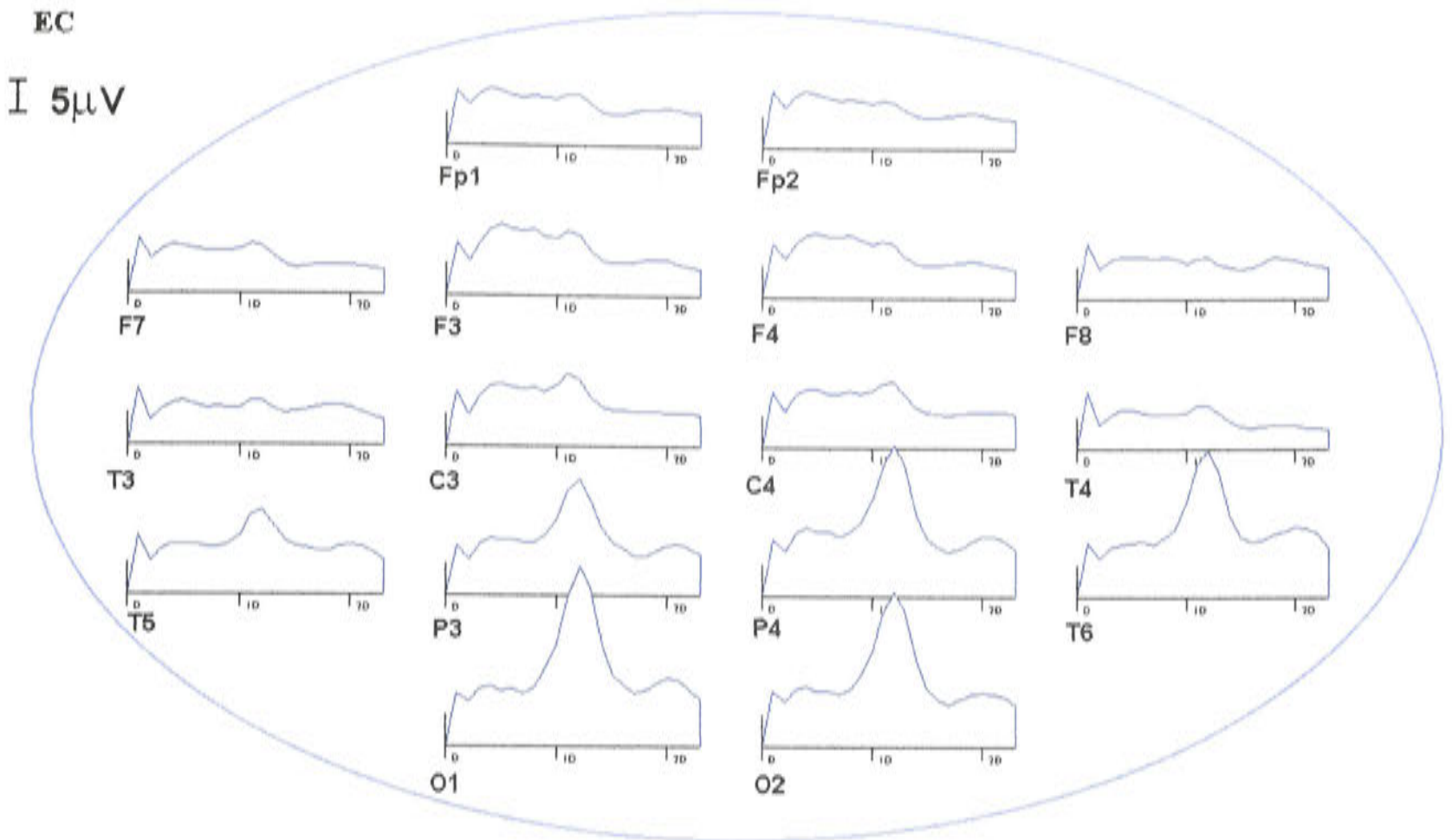



Figure 4.8a. Spectral plot of eyes closed EEG (μV) across 16 scalp sites (markers at 0, 10, & 20Hz) showing dominant occipital alpha frequency (11Hz) in 22year old, right handed, depressed female, BDI-II=41.

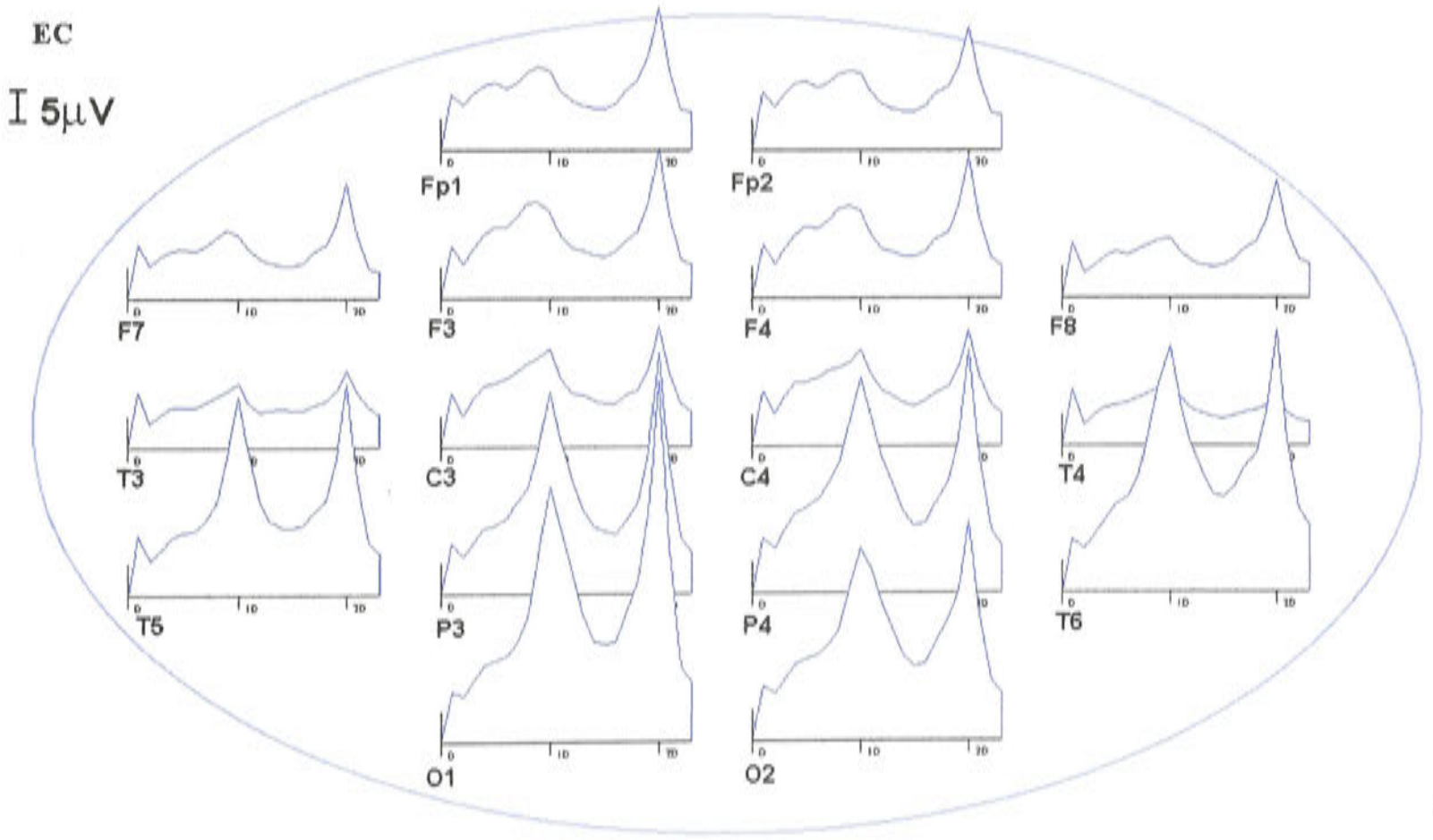



Figure 4.8b. Spectral plot showing 10Hz photic stimulation in eyes closed EEG (μV) across 16 scalp sites, with a 20Hz harmonic visible across the scalp, in the same 22yr old female.

Pockberger (1985) suggested that frequency following responses increase with increasing severity of depression and attenuate on recovery from depression. Pearson's correlation coefficients between depression level and frequency following response showed little to no relationship at baseline, $r = .05$, $p > .05$; at session 2, $r = -.12$, $p > .05$; session 3, $r = .03$, $p > .05$; or at follow-up $r = -.03$, $p > .05$.

Similar to Study 1 there was no evidence of entrainment to the three experimental photic stimulation frequencies (5Hz, 13Hz, and 22Hz). Unlike Study 1, however, the current study did not record EEG while participants were exposed to these frequencies, rather, it was reliant on finding evidence of 'a training effect' in eyes closed EEG after regular daily exposure to experimental photic stimulation frequencies. Separate three way MANOVA's, with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, session (1 to 4), and site (F3, F4, C3, C4, P3, P4, O1, and O2), as the within groups variables, were computed for each bandwidth, 5Hz, 13Hz, and 22Hz. It was anticipated that exposure to a particular photic stimulation frequency may increase EEG amplitude in that frequency band during the experimental phase and possibly also at follow-up, but not at baseline, therefore, only session by group interactions were of interest.

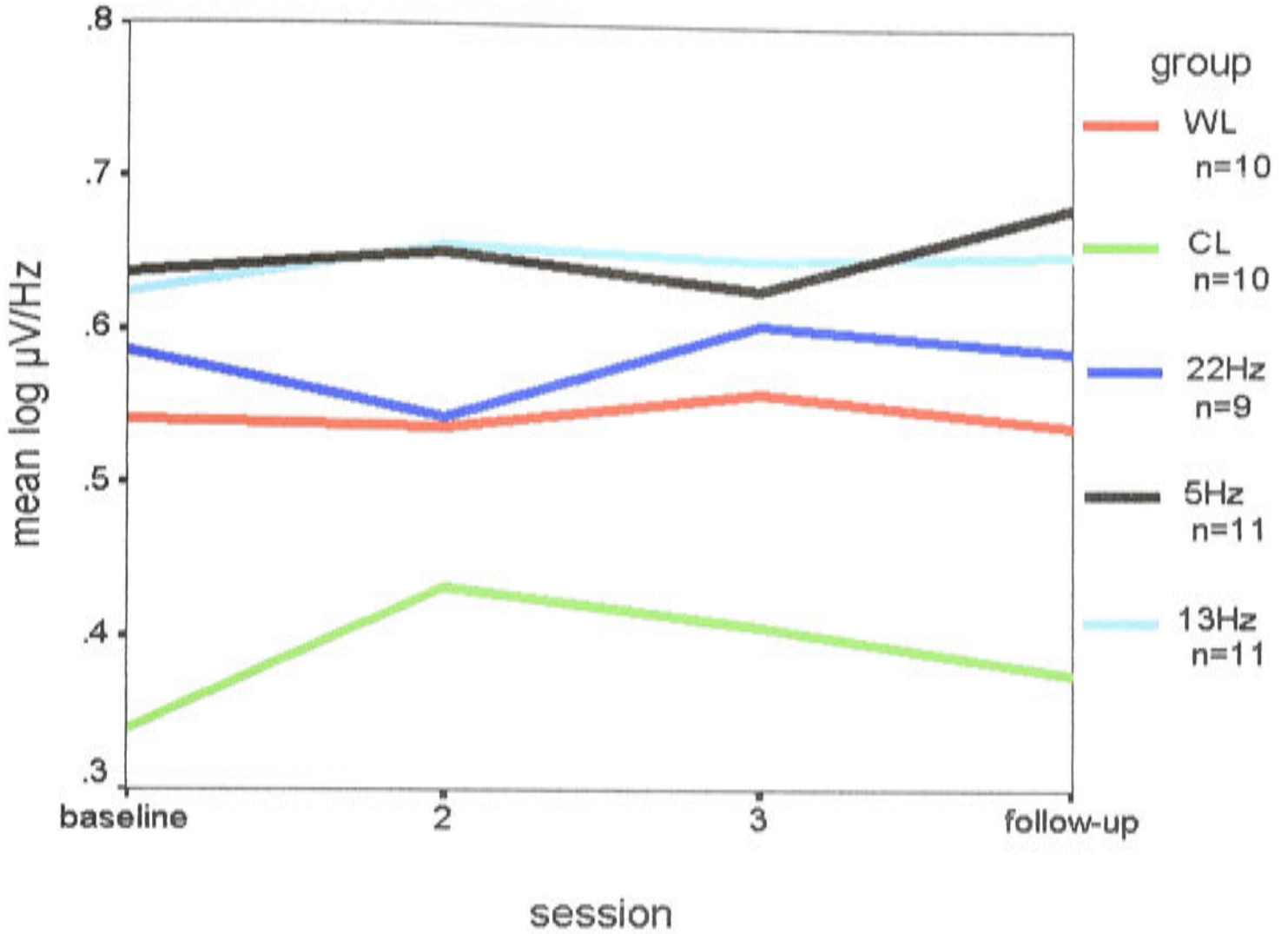



Figure 4.9 Mean log magnitude 13Hz in eyes closed EEG averaged across F3, F4, C3, C4, P3, P4, O1 and O2, for wait list (WL), continuous light (CL), 5Hz, 13Hz, and 22Hz groups.

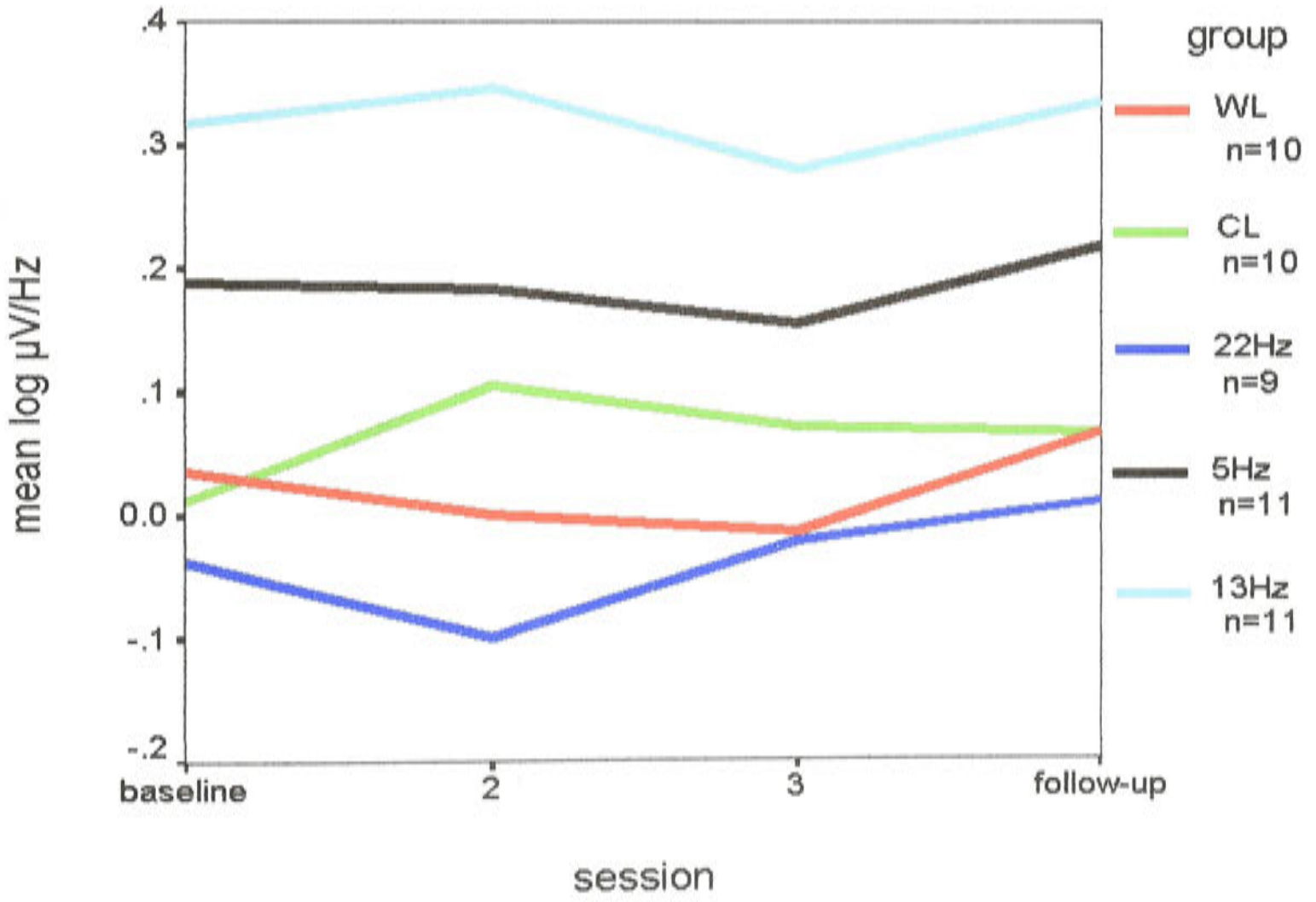



Figure 4.10 Mean log magnitude in 22Hz eyes closed EEG averaged across F3, F4, C3, C4, P3, P4, O1 and O2, for wait list (WL), continuous light (CL), 5Hz, 13Hz, and 22Hz groups.

As can be seen in Figures 4.9 and 4.10 there was little evidence of increased EEG magnitude for the 13Hz or 22Hz groups with a small drop in 22Hz magnitude for the 22Hz group after 2 weeks of light therapy and a gradual return to baseline at the end of the experimental phase. There was no change across sessions for the 13Hz group at their entrainment frequency.

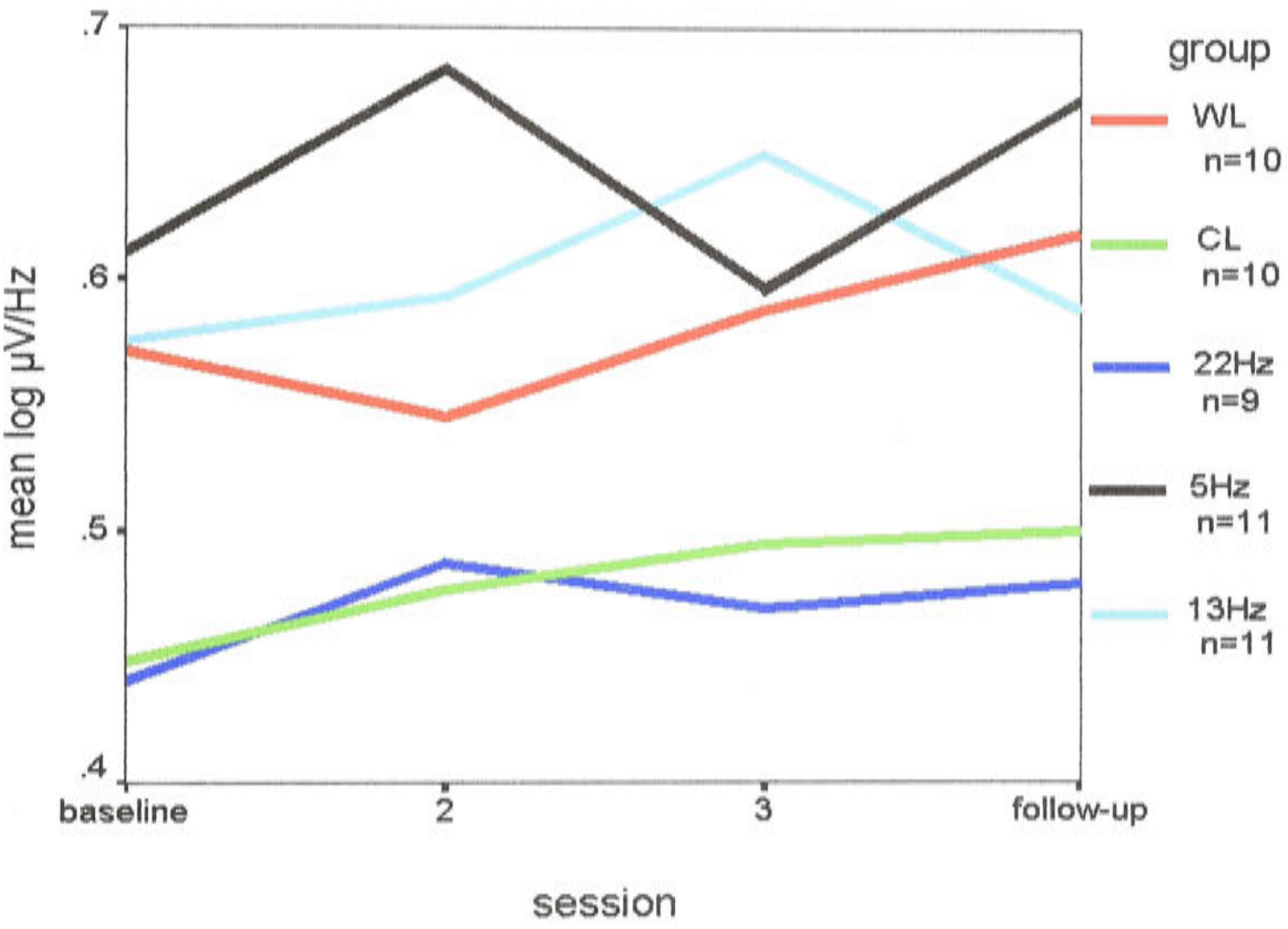



Figure 4.11 Mean log magnitude in 5Hz eyes closed EEG averaged across F3, F4, C3, C4, P3, P4, O1 and O2, for wait list (WL), continuous light (CL), 5Hz, 13Hz, and 22Hz groups.

The 5Hz group (see Figure 4.11) showed a slight increase in 5Hz magnitude after 2 weeks of light therapy, only to return to baseline levels by the end of the month. On analysis, session by group interaction effects were very small and non-significant for all experimental frequencies, 5Hz, $F(12,138) = 1.12$, $p > .05$, $\eta^2 = .09$; 13Hz, $F(12,138) = .66$, $p > .05$, $\eta^2 = .05$; and 22Hz, $F(12,138) = 1.23$, $p > .05$, $\eta^2 = .10$. In addition, there were no main effects for group or session. Thus there was no

evidence of increased EEG amplitude in these bandwidths that could be attributable to experimental manipulation.

Due to previous findings of increased positive affect with alpha AVS and decreased positive affect with beta AVS in Study 1, it was predicted that those receiving 13Hz photic stimulation would report higher levels of well-being than those receiving 22Hz photic stimulation immediately after light mask use. Figure 4.12 shows that all the active photic stimulation groups reported higher well-being after light therapy in comparison to the sham or continuous light group. This pattern was repeated with rated well-being prior to light therapy with no distinction evident between the three active photic stimulation frequencies. Contrary to predictions, the 22Hz group report higher well-being after photic stimulation than the 13Hz group.

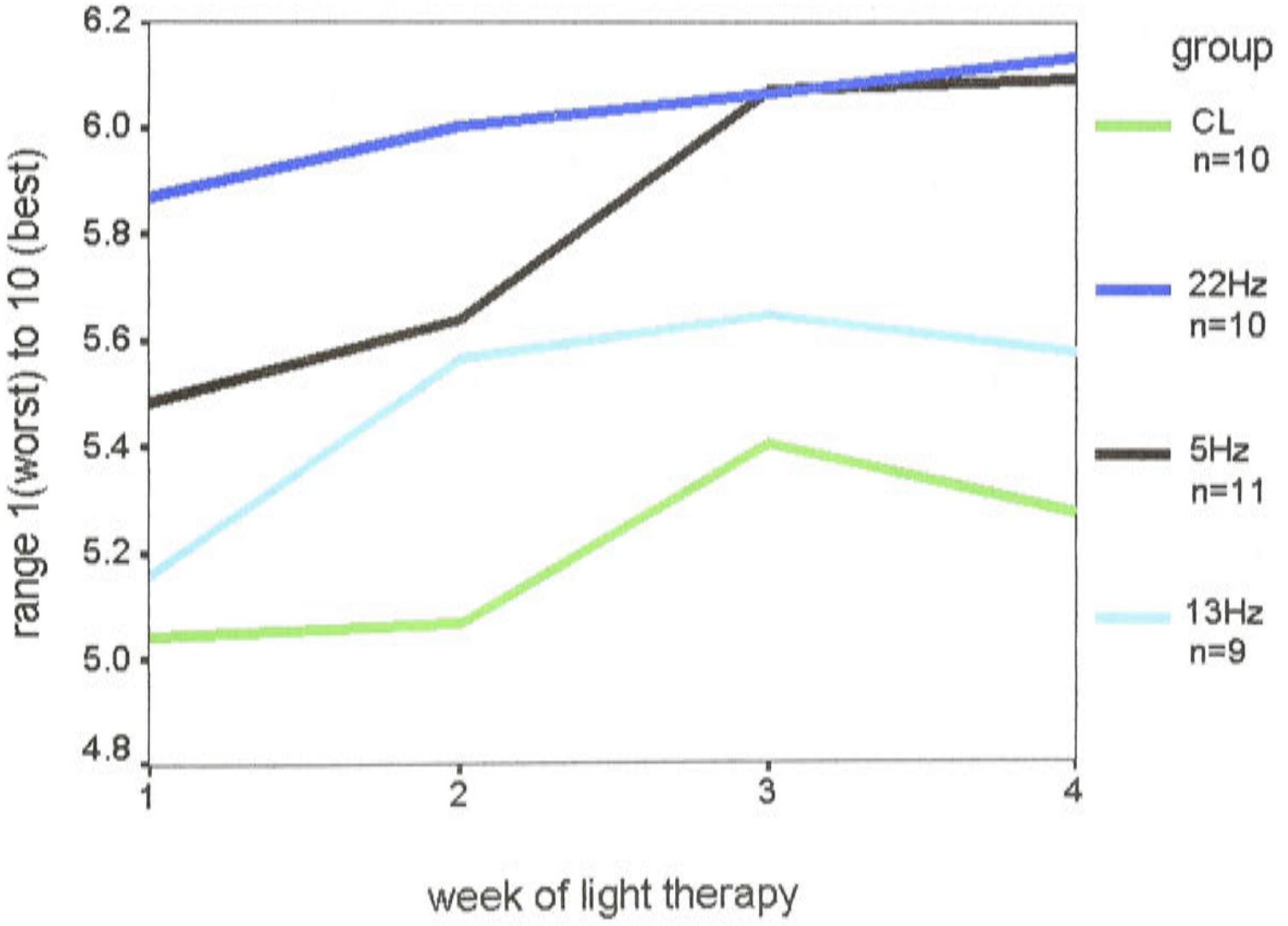



Figure 4.12 Mean well-being rating immediately after Lightmask™ use for photic stimulation groups; continuous light (CL), 22Hz, 5Hz, and 13Hz.

Omnibus three way MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and time (before and after) and session (1 to 4) as the within groups variables revealed a main effect for time, $F(1,36) = 20.60$, $p < .05$, $\eta^2 = .36$, with participants reporting enhanced well-being after light therapy, and a main effect for session, $F(3,34) = 3.90$, $p < .05$, $\eta^2 = .25$, indicating an increase in reported well-being across sessions. To test the hypothesis a time by group interaction was expected. This was not found, $F(3,36) = .88$, $p > .05$, $\eta^2 = .07$. There was no main effect for group, $F(3,36) = 1.20$, $p > .05$, $\eta^2 = .09$, suggesting that the observed differences between the active experimental photic stimulation conditions and the CL condition were not significant.

Time of day of light mask use was also recorded to assess its effect on mood and sleep parameters. As previously reported, most participants used their light masks between 9 and 10 pm (range 2pm to 12pm) before retiring for the night. There was a consistent negative correlation between time of light mask use and well-being after using the light mask, $r = -.13$, $p > .05$, for week 1; $r = -.25$, $p > .05$, for week2; $r = -.50$, $p < .05$, for week3; and $r = -.25$, $p > .05$, for week 4, possibly indicating a decrease in rated well-being with increasing tiredness with later light mask use.

Time of light mask use had little impact on depression levels, $r = .12$, $p > .05$, at week 2, and $r = .25$, $p > .05$, at week 4. Similarly, time of light mask use was unrelated to positive affect, $r = .08$, $p > .05$, at week 2, and $r = -.28$, $p > .05$ at week 4; and negative affect, $r = -.07$, $p > .05$, at week 2, and $r = .11$, $p > .05$, at week 4.

Later light mask use was associated with decreased sleep onset latency, $r = -.24$, $p > .05$, for week 1, $r = -.30$, $p < .05$, for week 2, $r = -.15$, $p > .05$, for week 3, and $r = .04$, $p > .05$, for week 4; but had no impact on night wakings, $r = .19$, $p > .05$, for week 1 $r = -.03$, $p > .05$, for week 2; $r = .08$, $p = .05$, for week 3; and $r = .09$, $p > .05$, for week 4; or sleep efficiency, $r = .13$, $p > .05$, for week 1, $r = .18$, $p > .05$, for week 2, $r = .08$, $p > .05$, for week 3, and $r = .03$, $p > .05$, for week 4.

There was little variation in the time of day light masks were used with most participants adhering to study instructions and using their light masks in the evening. Consequently, there was little correlation between time of day of light mask use and sleep and mood parameters. In addition, it is difficult to tease out the impact of increasing fatigue, which invariably exerts its effects on mood and sleep parameters, such as sleep onset, as the day progresses.

**4.10.5 Subjective relaxation**: It was predicted that those receiving 5Hz and 13Hz photic stimulation would report higher subjective relaxation during the experimental phase than those in the 22Hz, CL or WL groups. This hypothesis was tested using a two way MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable and session (1-4) as the within groups variable. As can be seen in Figure 4.13 the hypothesis was only partially supported with the 5Hz group showing the highest subjective relaxation across sessions, and the 22Hz group reporting the lowest subjective relaxation ratings. This observed trend was not supported statistically with no session by group interaction present, $F(12, 141) = 0.81$, $p > .05$, $\eta^2 = .06$. It can also be seen that subjective relaxation was variable for all groups with no clear trends observed across sessions. This was indicated by a very small

non-significant session effect, $F(3,45) = .73$, $p > .05$, $\eta^2 = .06$. Finally, there was no main effect for group, $F(4,47) = 1.23$, $p > .05$, $\eta^2 = .09$.

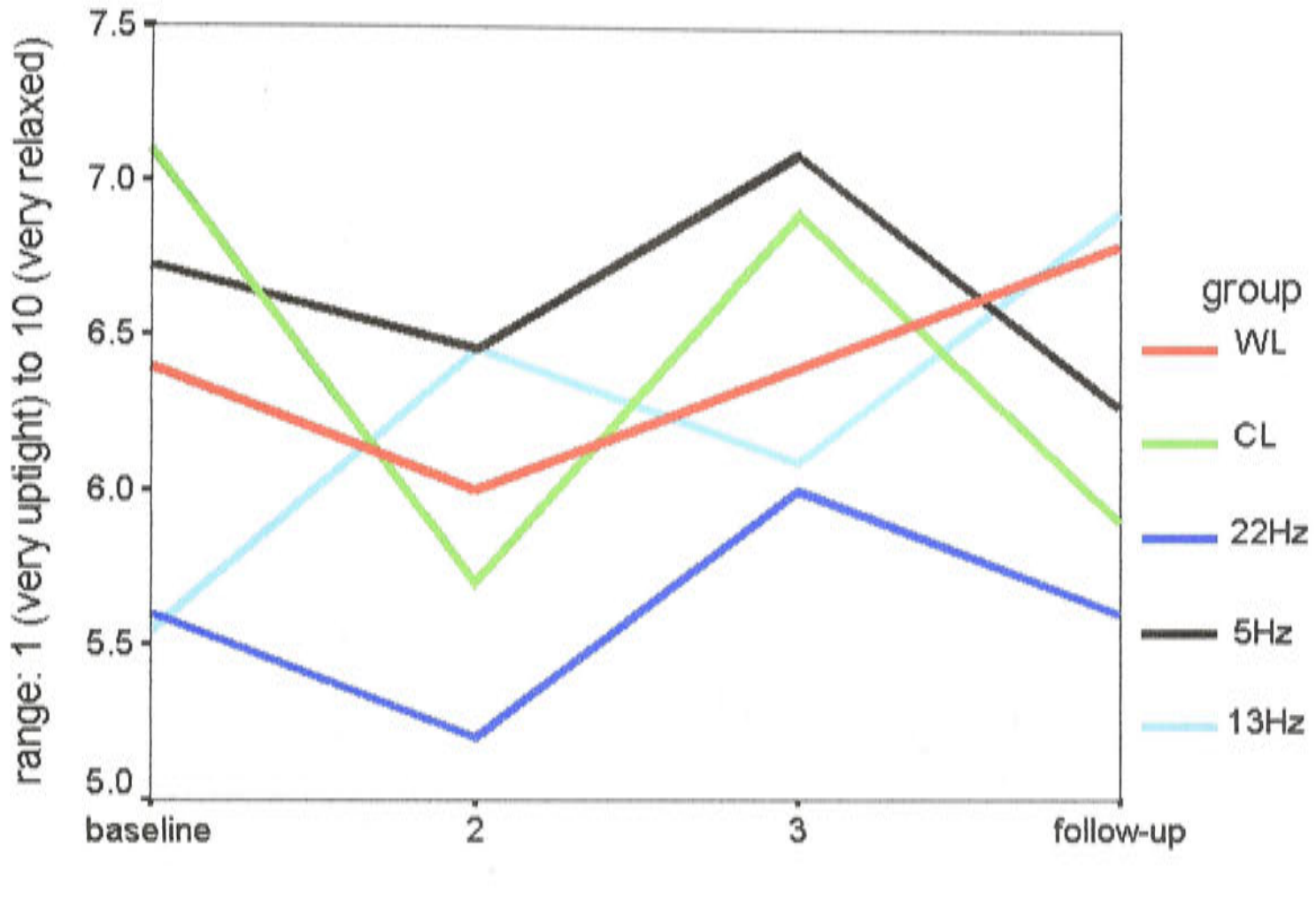



Figure 4.13 Mean subjective relaxation across sessions.

In addition, it was expected that subjective relaxation would correlate positively with theta and alpha, and negatively with beta frequencies across sessions. Contrary to expectations small to moderate inverse correlations were found between subjective relaxation and theta at different sites, $r = -.12$ to $-20$, $p > .05$, and alpha, $r = -.39$, $p < .05$, to $-.25$, $p > .05$ at baseline. A trend in predicted directions was found between subjective relaxation and beta amplitude with small inverse correlations revealed, $r = -.25$ to $-.13$, $p > .05$. This pattern of relationship between beta and subjective relaxation was not repeated at session 2, with near zero correlations revealed for all

bandwidths across the scalp, but was repeated again at session 3, r = -.14 to -.11, p > .05, and was accompanied by near zero correlations between theta and alpha across the scalp. At follow-up, higher subjective relaxation was associated with decreased beta amplitude across frontal sites, r = -.31, p < .05, midline, r = -.34, p < .05, and posteriorally, r = -.14, p > .05, but no relationship between subjective relaxation and alpha or theta. Thus subjective relaxation tended to be associated with a decrease in beta amplitude rather than increases in theta or alpha, particularly in frontal and midline regions rather than posterior regions.

**4.10.6 Mood (PANAS):** As discussed in Chapter 2, positive and negative affect are purported to be orthogonal constructs (Diener & Emmons, 1985; Watson, Clark, & Tellegen, 1988). Prior to use in the current study, the factor structure of the PANAS was assessed using visual observation of the scree plots and factor analysis. The orthogonal structure was supported at some administrations, but not at others, with two strong main factors generally present, but at times a possible weaker third factor also present.

Specifically, factor analysis of PANAS items revealed a strong positive affect factor and a less robust negative affect factor on all administrations except at session two. On the second administration of the PANAS positive affect was less robust with three items, 'excited', 'proud', and 'alert' loading on the negative affect factor. Despite this, Pearson's correlation coefficients between positive affect and negative affect at session two were small and non-significant, r = .14, p > .05). Consistent with the independence hypothesis for these two constructs, there was no relationship between positive affect and negative at session 1, r = .04, p > .05; session 3, r =-.09, p > .05;

nor at follow-up, r =-.24, p > .05. These findings were similar to Study 1. The alpha reliability coefficients for the two scales in the current study ranged from .87 to .92 for positive affect and .86 to .89 for negative affect across administrations, therefore all items were retained for analysis.

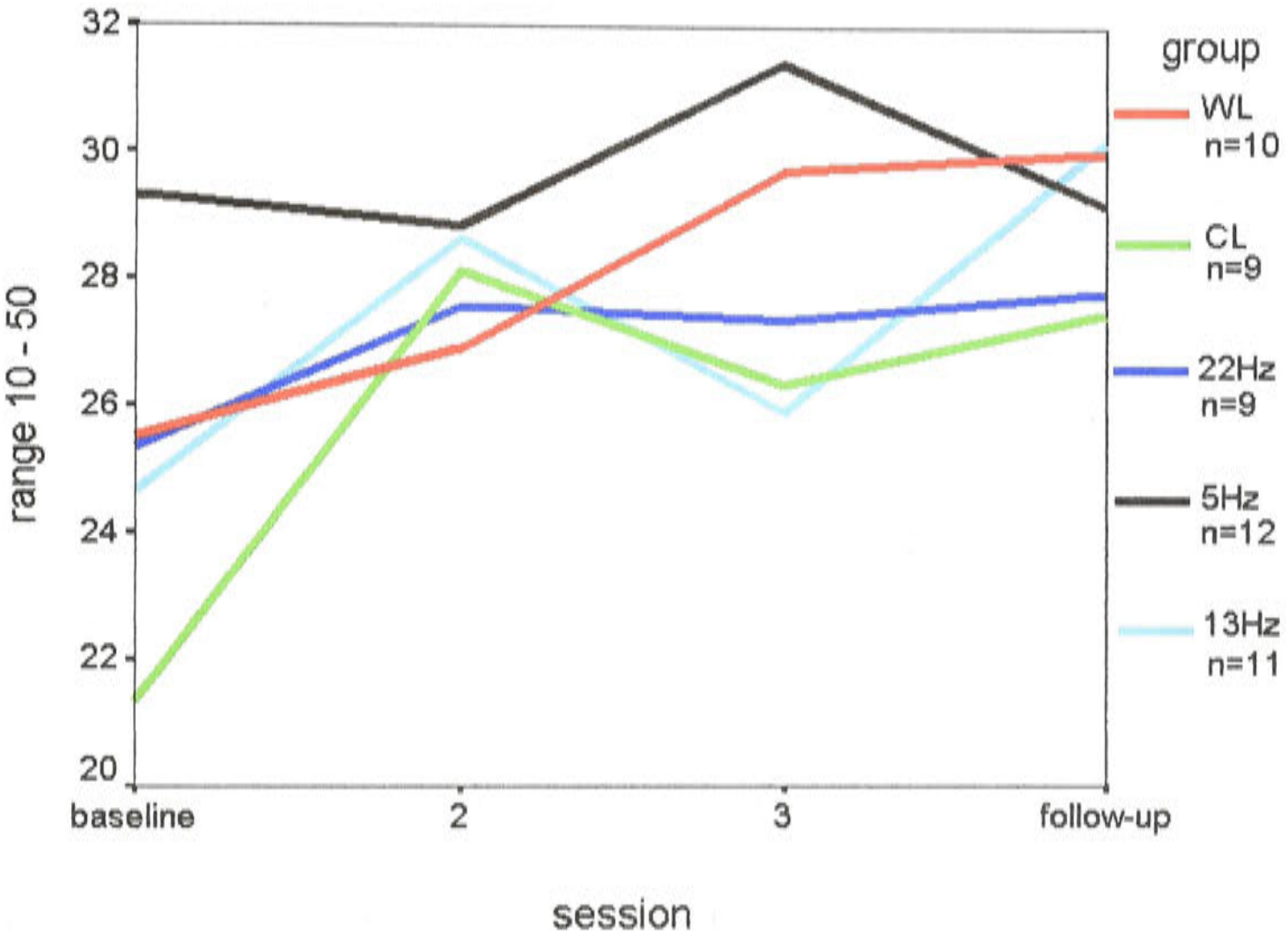



Figure 4.14 Mean positive affect (PANAS) across sessions.

It was hypothesised that active photic stimulation would induce a relaxation response and produce greater positive affect and lower negative affect in the 5Hz, 13Hz, and 22Hz groups in comparison to the WL or CL. This was tested using three way MANOVA's with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and session (1 to 4) and items (1-10) as the within groups variables for both positive and negative affect. Unlike Study 1, positive affect increased for all groups

over sessions with a significant main effect for session, $F(3,44) = 3.91$, p,.05, $\eta^2 = .21$ revealed (see Figure 4.14). This effect, however, was independent of group membership, $F(12,138) = .93$, $p > .05$, $\eta^2 = .07$. Particular adjectives, such as 'interested' and 'alert' were endorsed more than other positive affect adjectives with a main effect for items revealed, $F(9,38) = 20.18$, $p < .05$, $\eta^2 = .83$, but this was not dependent of group membership, $F(36,164) = .99$, $p > .05$, $\eta^2 = .18$. In addition, there was no main effect for group, $F(4, 46) = .63$, $p > .05$, $\eta^2 = .05$. The expected pattern of results was not found, the active photic stimulation groups did not report higher positive affect than the WL or CL groups as evidenced by a non-significant small session by group interaction effect, $F(12,138) = .93$, $p > .05$, $\eta^2 = .07$.

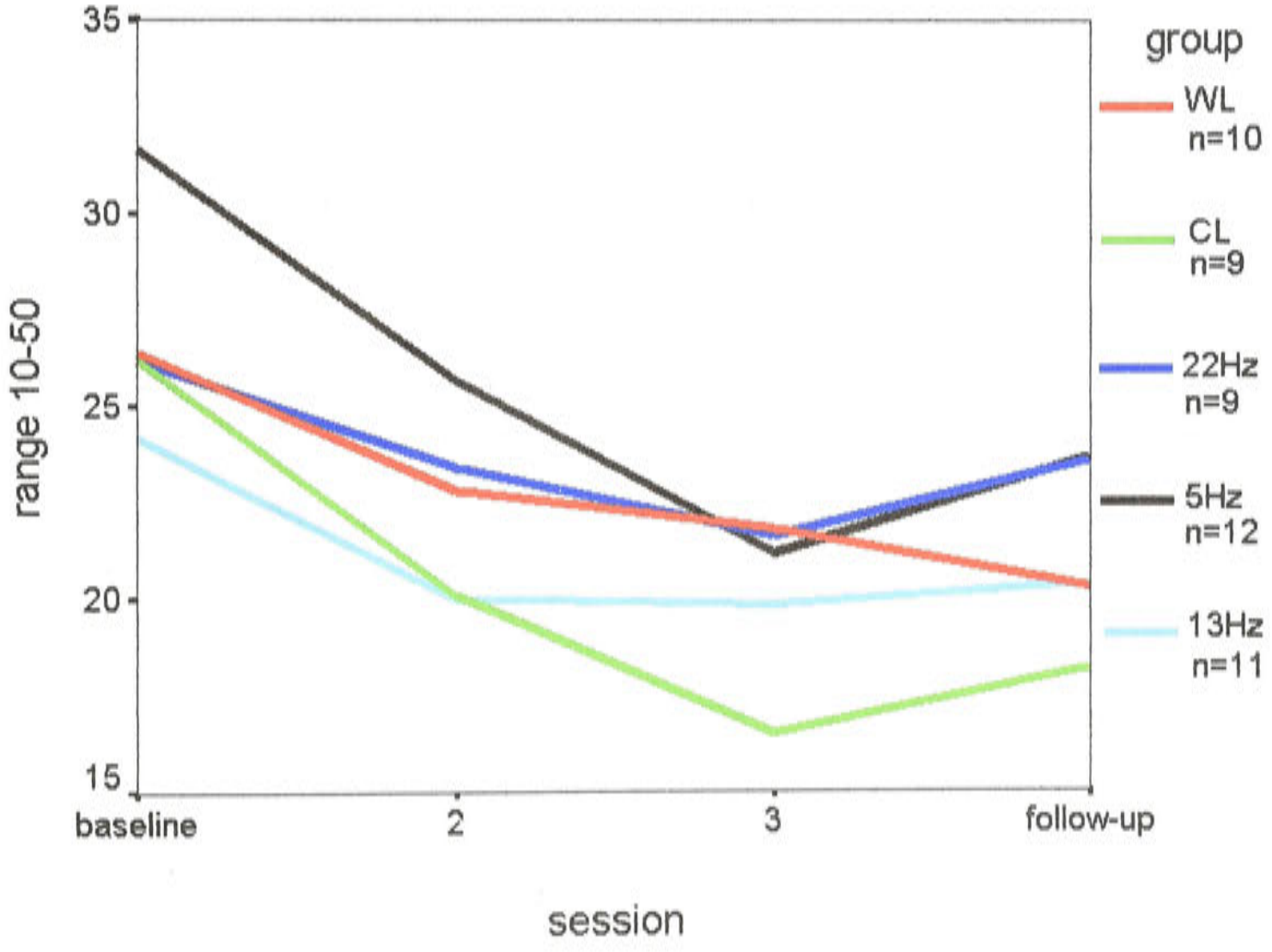



Figure 4.15 Mean negative affect (PANAS) across sessions.

As can be seen in Figure 4.15 negative affect declined over sessions for all participants. This was supported with a moderately strong main effect for session, $F(3,46) = 10.66$, $p < .05$, $\eta^2 = .41$. Further exploration of this effect revealed that negative affect was significantly lower than baseline measures at every subsequent session. The expected pattern of effects with the 5Hz, 13Hz, and the 22Hz groups showing greater reductions in negative affect than the WL or CL groups was not found as evidenced by a very small non significant session by group interaction effect, $F(12,144) = .56$, $p > .05$, $\eta^2 = .04$. In addition, there was no main effect for group, $F(4,48) = 1.31$, $p > .05$, $\eta^2 = .09$. Commensurate with Study 1, there was a main effect for items with participants endorsing adjectives such as 'irritable', 'upset', and 'distressed', more than other adjectives, but this was not dependent on group membership, $F(36,172) = 1.16$, $p > .05$, $\eta^2 = .19$.

**4.10.7 Depression:** Because of the relaxing effects of photic stimulation coupled with expectancy effects for all participants, it was anticipated that depression levels would decrease for all groups, with the greatest amount of decrease observed in those receiving active light therapy (5Hz, 13Hz or 22Hz). This prediction was tested using a two way MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and session (1 to 4) as the within groups variable. There were no differences between the groups at baseline. As can been seen in Figure 4.16, depression levels decreased for all groups across sessions as predicted. This was supported with a strong main effect for session, $F(3,45) = 36$, $p < .05$, $\eta^2 = .70$, with significant decreases in depression scores observed at every session in comparison to baseline. However, contrary to predictions, there was no difference between the active photic stimulation groups and the wait list and control groups during the

intervention phase, as evidenced by a non significant session by group interaction, $F(12, 141) = 1.44$, $p > .05$, $\eta^2 = .11$. There was no main effect for group, $F(4,47) = .34$, $p > .05$, $\eta^2 = .03$.

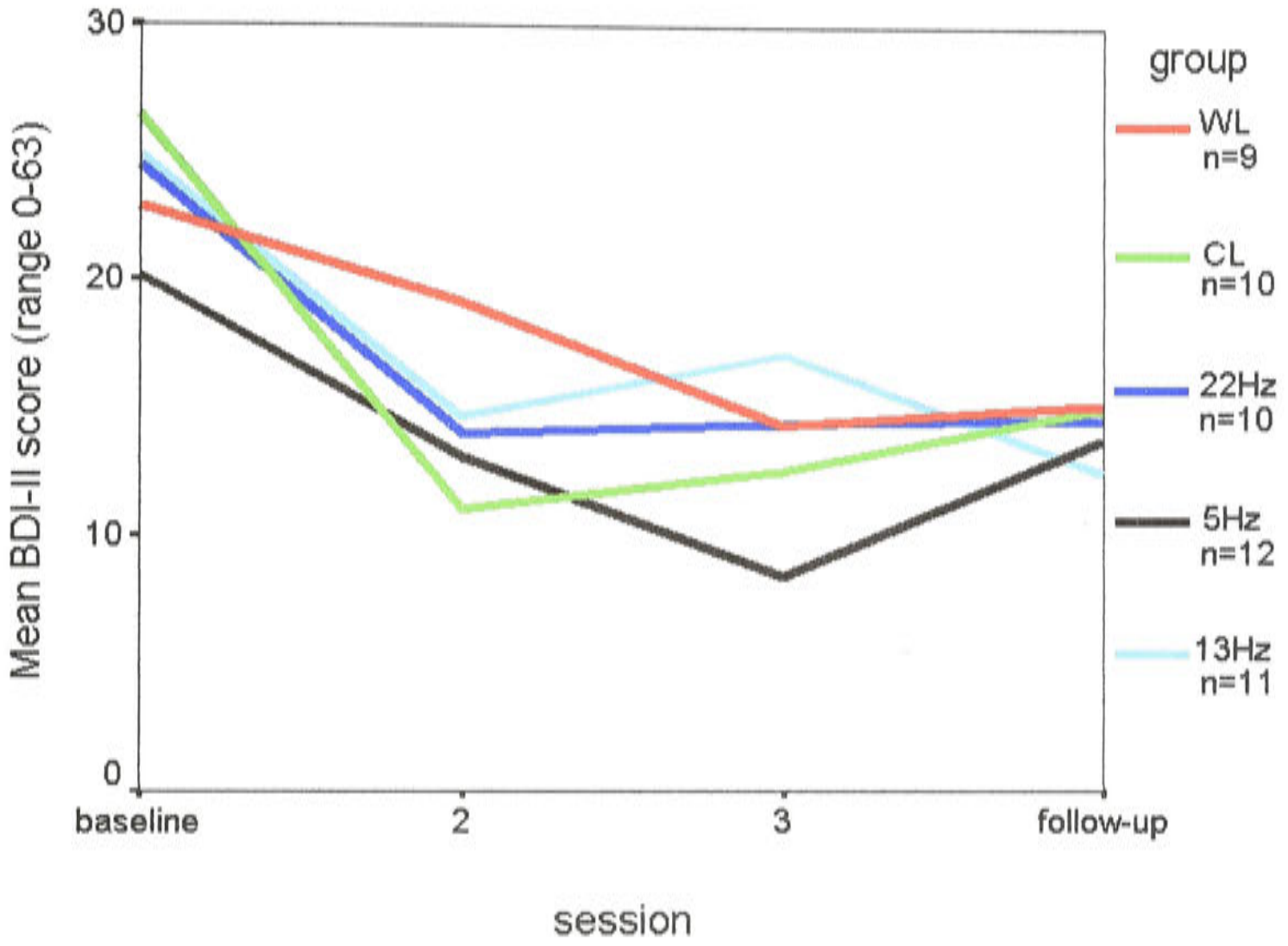



Figure 4.16 Mean depression level (BDI-II) across sessions.

**4.10.8 Anxiety:** It was also hypothesised that anxiety levels would decrease over sessions as relaxation responses became realised. In particular it was predicted that 5Hz photic stimulation would most benefit those with high anxiety levels as it would assist in reducing possible high beta which can be associated with high anxiety, and help to reduce hyper arousal. Whereas, photic stimulation at 22Hz was expected to benefit those with low anxiety as it would assist in increasing motivation and alertness with the generation of higher cortical activity. There were no differences

between groups in anxiety level at baseline. Omnibus MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and session (1 to 2) as the within groups variable revealed a moderate main effect for session, $F(1,49) = 39.62$, $\eta^2 = .46$, but no session by group interaction, $F(4,49) = 1.12$, $\eta^2 = .08$. There was no main effect for group, $F(4,49) = 1.50$, $\eta^2 = .12$.

Table 4.1 Mean SCL-90-R anxiety T-Scores at baseline and follow-up for low (< 64) and high(≥ 64) anxiety participants.

| | Baseline | | Follow-up | |
|---|---|---|---|---|
| | low | high | low | high |
| Group | Mean (SD) | Mean (SD) | Mean (SD) | Mean (SD) |
| WL | 49.67 (6.98) (n=6) | 68.25 (5.44) (n=4) | 45.50 (6.12) | 61.25 (8.77) |
| CL | 58.33 (1.15) (n=3) | 71.71 (6.63) (n=7) | 45.67 (7.77) | 61.71 (7.25) |
| 22Hz | 54.75 (11.98) (n=4) | 72.17 (5.64) (n=6) | 47.75 (9.18) | 68.67 (10.37) |
| 5Hz | 55.67 (6.51) (n=3) | 72.44 (6.84) (n=9) | 53.67 (8.96) | 64.11 (8.96) |
| 13Hz | 56.14 (5.40) (n=7) | 70.80 (6.42) (n=5) | 50.14 (10.99) | 67.40 (13.67) |

WL= wait list, CL = continuous light.

To assess this hypothesis more fully, separate two way MANOVA's with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and session (1 to 2) as the within groups variable were computed for high anxiety and low anxiety participants using a median split for anxiety. Effectively this split the sample at a T-score of 64 dividing the sample into low and high anxiety. This was a convenient split as a T-score of 63 or higher signifies a person 'at risk' of pathology according to Derogatis, (1977). There was a main effect for session for both low and high anxiety participants, $F(1,18) = 14.48$, $p < .05$, $\eta^2 = .45$, and $F(1,26) = 19.10$, $p < .05$, $\eta^2 = .42$, respectively, but no predicted session by group interactions for low anxiety, $F(4,18)$

= .90, $p > .05$, $\eta^2 = .17$, or for high anxiety, $F(4,26) = .86$, $p > .05$, $\eta^2 = .12$ (see Table 4.1 for means).

**4.10.9 Symptom severity**: Symptom severity (Global Severity Index SCL-90-R) was predicted to decrease more for the active photic stimulation groups in comparison to WL or CL across sessions, because of the arousal reducing ability of photic stimulation. This hypothesis was tested using a two way MANOVA with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and session (1 to 2) as the within groups variable. As can be seen in Table 4.2, global severity index T-scores decreased significantly for all groups across sessions as evidenced by a main effect for session, $F(1,49) = 48.97$, $p < .05$, $\eta^2 = .50$. However, the hypothesis was not supported, with the WL and CL groups demonstrating similar decreases in symptom severity to the active photic stimulation groups at follow-up with a non-significant session by group interaction revealed, $F(4,49) = .48$, $p > .05$, $\eta^2 = .04$. There was no main effect for group $F(4,49) = .65$, $p > .05$, $\eta^2 = .05$.

Table 4.2 Mean SCL-90-R global severity index T-Scores at baseline & follow-up

| | Baseline | Follow-up |
|---|---|---|
| | Mean (SD) | Mean (SD) |
| WL (n=6) | 64.30 (9.26) | 59.40 (9.47) |
| CL (n=3) | 69.90 (10.04) | 62.70 (13.30) |
| 22Hz (n=4) | 68.50 (10.26) | 61.90 (14.52) |
| 5Hz (n=3) | 70.33 (7.24) | 64.83 (8.13) |
| 13Hz (n=7) | 67.17 (6.78) | 58.67 (12.44) |

WL= wait list, CL = continuous light.

Contrary to reports, there was little to no correlation between anxiety and EEG measures across sessions for either local broadband EEG measures or global measures, r =.01, p > .05, for global theta; r = -.19 to -.11, p > .05 for global alpha; and r = -.08 to -.14, p > .05 for global beta. Thus the hypothesised trend, that higher anxiety levels would be associated with higher beta amplitudes was not found. Rather, correlations between anxiety and localised broadband EEG measures for each of the sixteen sites, tended to be small and inverse (r~-.20, p > .05), suggesting that anxiety was associated with a decrease in amplitude across all bandwidths.

Likewise for depression, there was little to no correlation between depression and either local broadband or global EEG measures across sessions, with a tendency for EEG amplitude in all bandwidths to decrease as depression level increased, r = .02 to -.19, p > .05, for global theta; r =-.19 to .05, p > .05, for global alpha, and r = -.13 to .05, p > .05, for global beta.

Depression and anxiety correlated moderately together both at baseline, r = .51, p < .01, and follow-up, r = .66, p < .01. Consistent with Watson and Clark (1988), depression was moderately negatively correlated with positive affect, r =-.59, to -.34, p < .01, across the four sessions, and positively correlated with negative affect, r = .41 to .61, p < .01, indicating that depression comprised negative affect with an absence of positive affect. Anxiety, on the other hand, was unrelated to positive affect, r = -.22 to -.14, p > .05, and strongly related to negative affect, r = .58 to .69, p < .01, across sessions. In addition, symptom severity correlated strongly with both depression, r = .78 to .80, p < .05, and anxiety, r = .70 to .80, p < .05.

**4.10.10 Sleep and photic stimulation:** At baseline, 62% of participants reported initial difficulty falling asleep, 72% reported difficulty falling asleep after waking during the night, and 58% reported waking during the night and lying awake worrying. Only 30% of participants reported night sweats and nightmares, which are symptoms consistent with high anxiety states. Most participants, 88%, reported waking feeling tired and 65% said they never quite got enough sleep. The majority of participants, 73%, reported sleep problems most nights while 20% reported experiencing sleep problems 'every other night'.

It was predicted that photic stimulation at 5Hz, 13Hz, or 22Hz would improve sleep parameters over and above no therapy or continuous light. In particular, it was proposed that photic stimulation at 5Hz would be most effective for reducing sleep onset latency, while photic stimulation at 13Hz was expected to offer the best treatment for night wakings. Separate MANOVA's were computed with group (WL, CL, 5Hz, 13Hz, and 22Hz) as the between groups variable, and time (week 1 to 6) as the within groups variable for sleep onset, night wakings, sleep efficiency and waking mood.

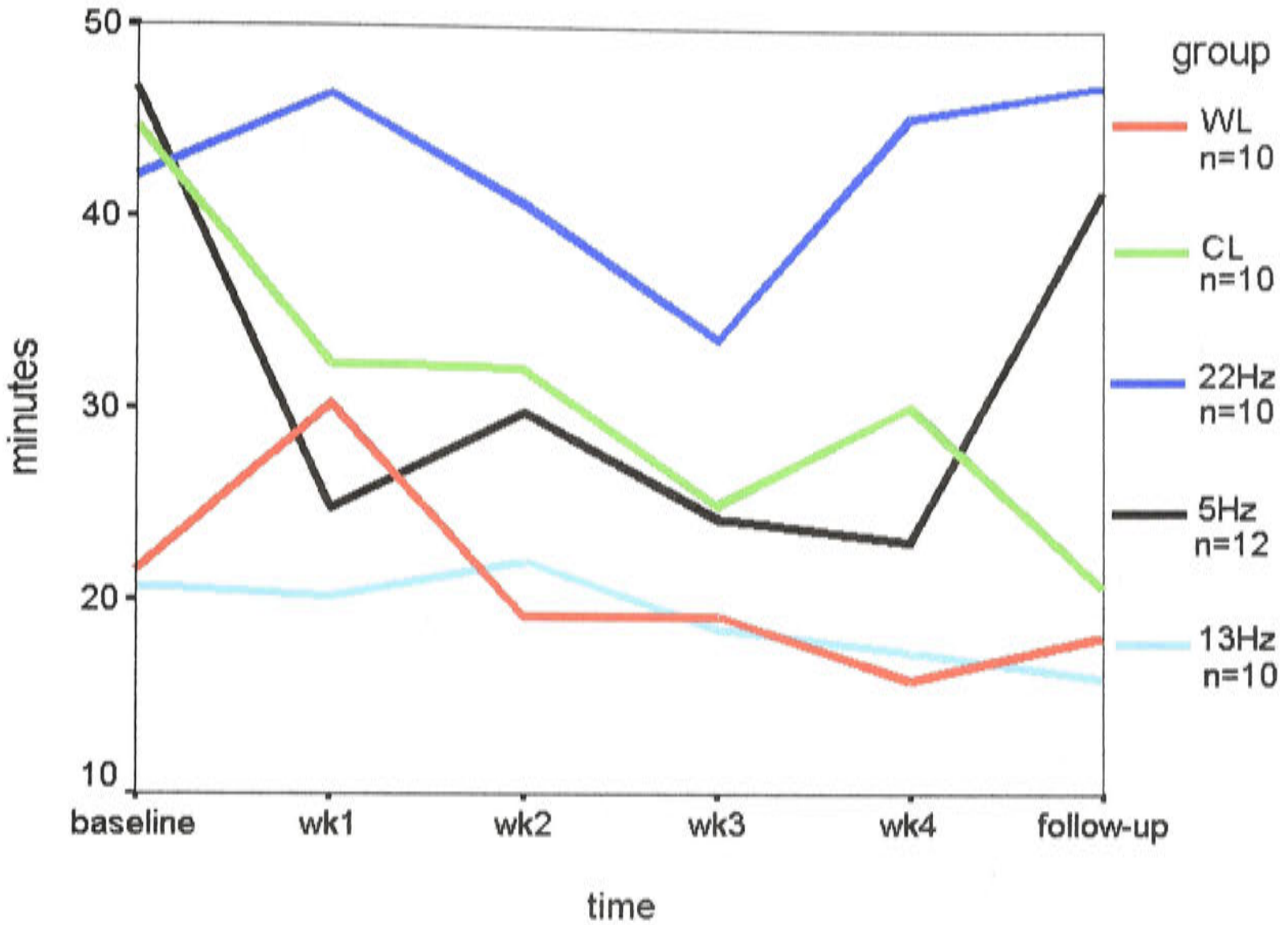



Figure 4.17 Mean 'sleep onset latency' across six weeks of sleep diary.

As can be seen in Figure 4.17, the WL and 13Hz groups displayed lower sleep onset latencies than the CL, 5Hz, and 22Hz groups at baseline, however these differences were not statistically significant. While sleep onset latency appears to decrease to a small extent over sessions, regardless of group membership, this effect was not significant, indicating that sleep onset latency remained relatively unchanged over the study period, $F(5,43) = 1.74$, $p > .05$, $\eta^2 = .17$. Consistent with predictions, the 22Hz group experienced higher sleep onset latencies than the 5Hz and 13Hz groups, but not lower sleep onset latencies than the WL or CL groups. These effects were small and non-significant, as evidenced by a non-significant session by group interaction, $F(20,184) = .98$, $p > .05$, $\eta^2 = .09$. There was no main effect for group, $F(4,47) = .63$, $p > .05$, $\eta^2 = .05$.

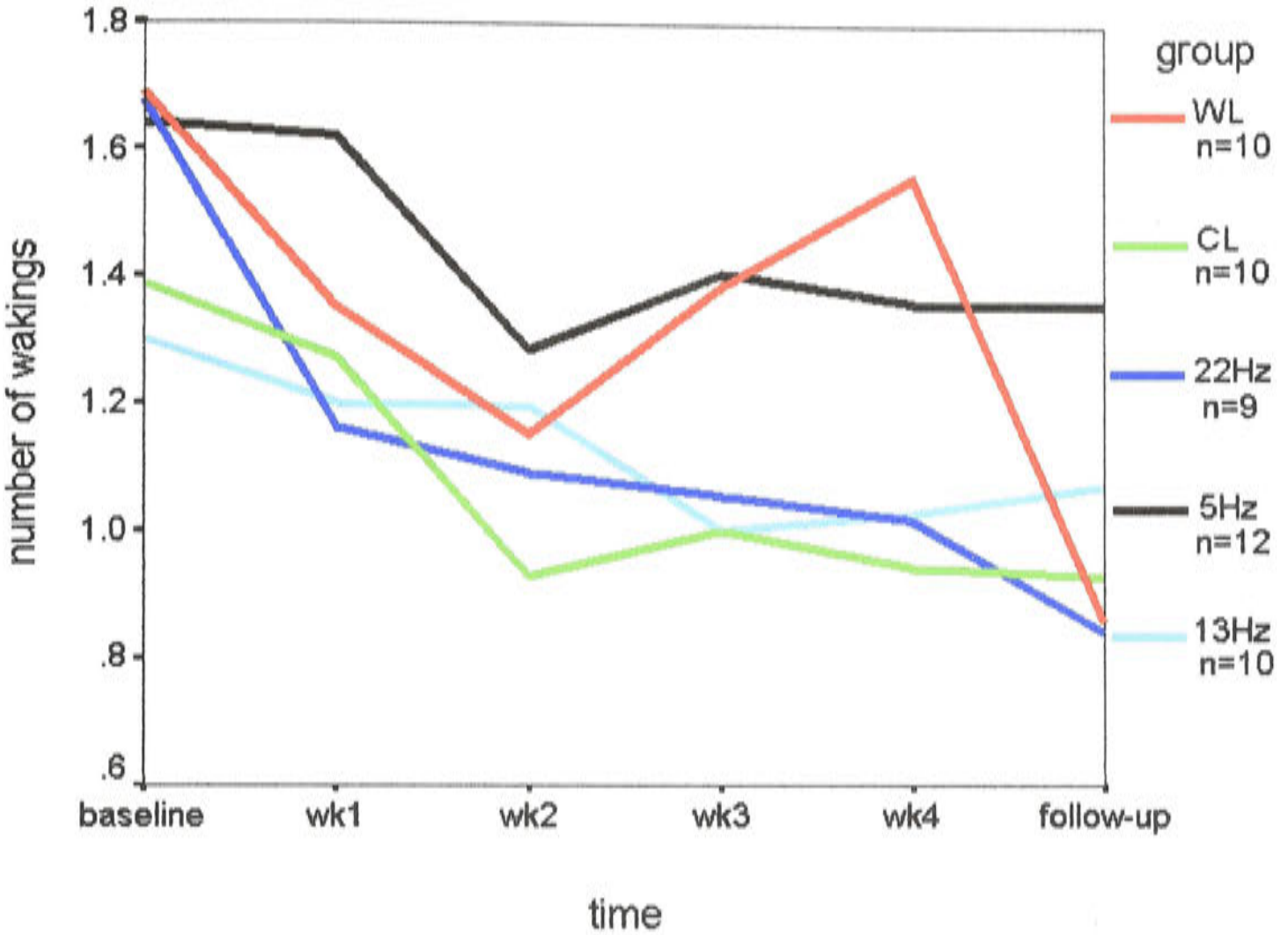



Figure 4.18 Mean number of 'night wakings' across six weeks of sleep diary.

Figure 4.18 shows that the average number of night wakings decreased for all groups in the first few weeks of the study with the WL group showing an increase in night wakings during weeks 3 and 4. This trend was supported with a significant main effect for time, $F(5,42) = 2.55$, $p < .05$, $\eta^2 = .23$. Further exploration of this effect found that the mean number of night wakings was significantly less at every week except week 2 in comparison to baseline. This decrease in night wakings over time was not dependent on group membership as proposed, with 13Hz photic stimulation being just as effective as photic stimulation at other frequencies as revealed by a non-significant session by group interaction, $F(20,180) = .68$, $p > .05$, $\eta^2 = .07$. There was no main effect for group, $F(4,46) = .32$, $p > .05$, $\eta^2 = .03$.

It was also hypothesised that photic stimulation at 5Hz, 13Hz and 22Hz would be more effective at improving sleep efficiency in comparison to WL or CL. As can be seen in Figure 4.19 there was a general trend of improvement in sleep efficiency for all groups over the course of the study. This was supported with a main effect for time, $F(5,43) = 3.21$, $p < .05$, $\eta^2 = .27$, with significant increases in sleep efficiency at every week post baseline. The expected pattern of effects, with the 5Hz, 13Hz and 22Hz groups showing greater improvement in sleep efficiency during the experimental phase, was not supported with a small non-significant time by group interaction effect revealed, $F(20,184) = .93$, $p > .05$, $\eta^2 = .09$. There was no main effect for group, $F(4,47) = 1.12$, $p > .05$, $\eta^2 = .09$.

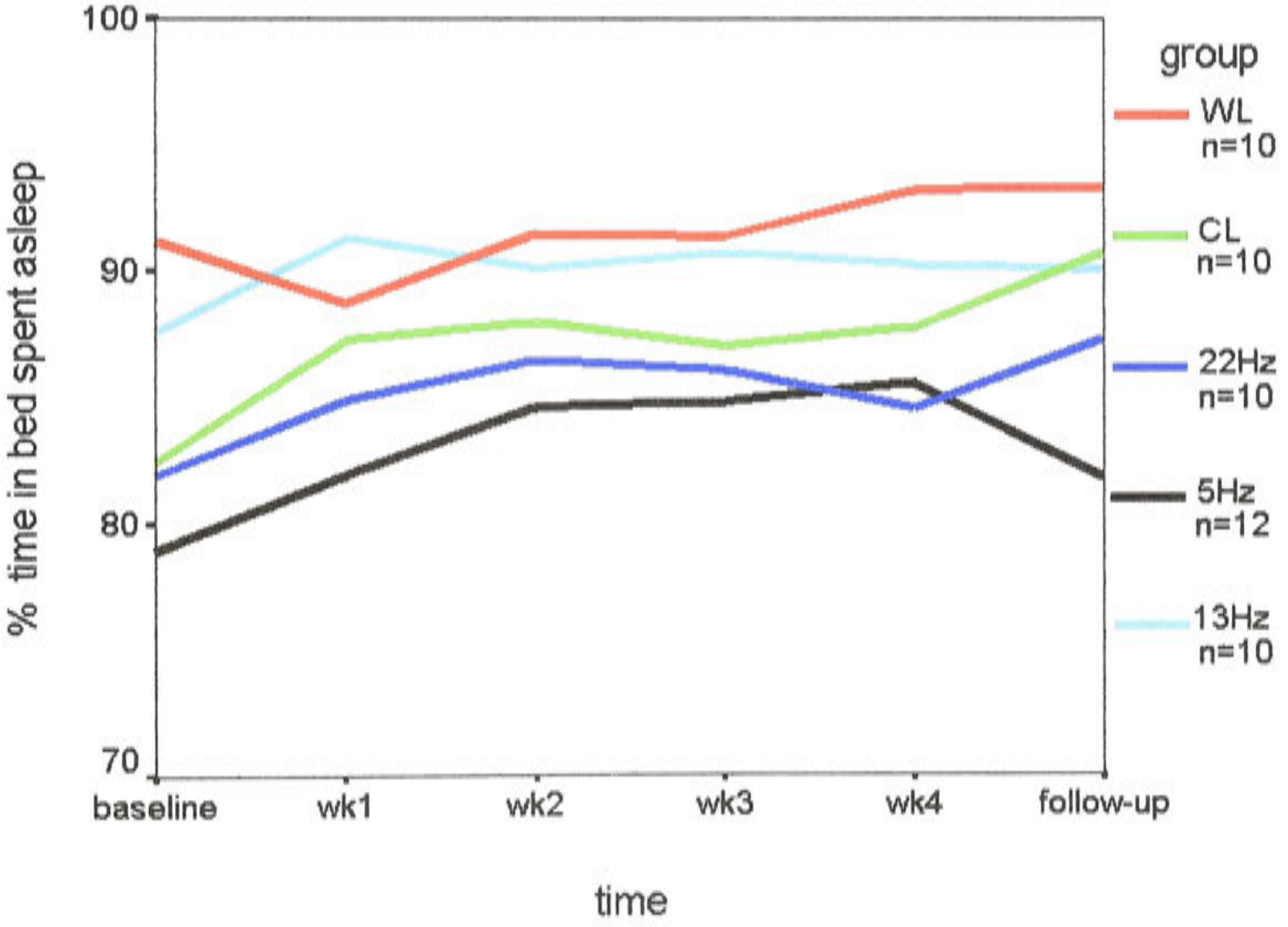



Figure 4.19 Mean sleep efficiency across six weeks of sleep diary.

It was predicted that those in the 5Hz, 13Hz, and 22Hz groups would report higher waking mood than those in the WL, or CL groups because of expected reductions in sleep onset latencies and night wakings for these groups. As can be seen in Figure 4.20, there were no differences between groups in waking mood at baseline with an increase in waking mood for all groups over the six weeks of sleep diary maintenance. This was supported with a significant main effect for time, $F(5,43) = 5.70$, $p < .05$, $\eta^2 = .40$, and characterised by significant increases in waking mood for every time period in comparison to baseline. The hypothesised pattern of effects was only partially supported, with all active photic stimulation groups reporting higher waking mood than those in the WL or CL groups. This effect was not significant, however, as evidenced by a non-significant time by group interaction effect, $F(20,184) = .54$, $p > .05$, $\eta^2 = .06$.

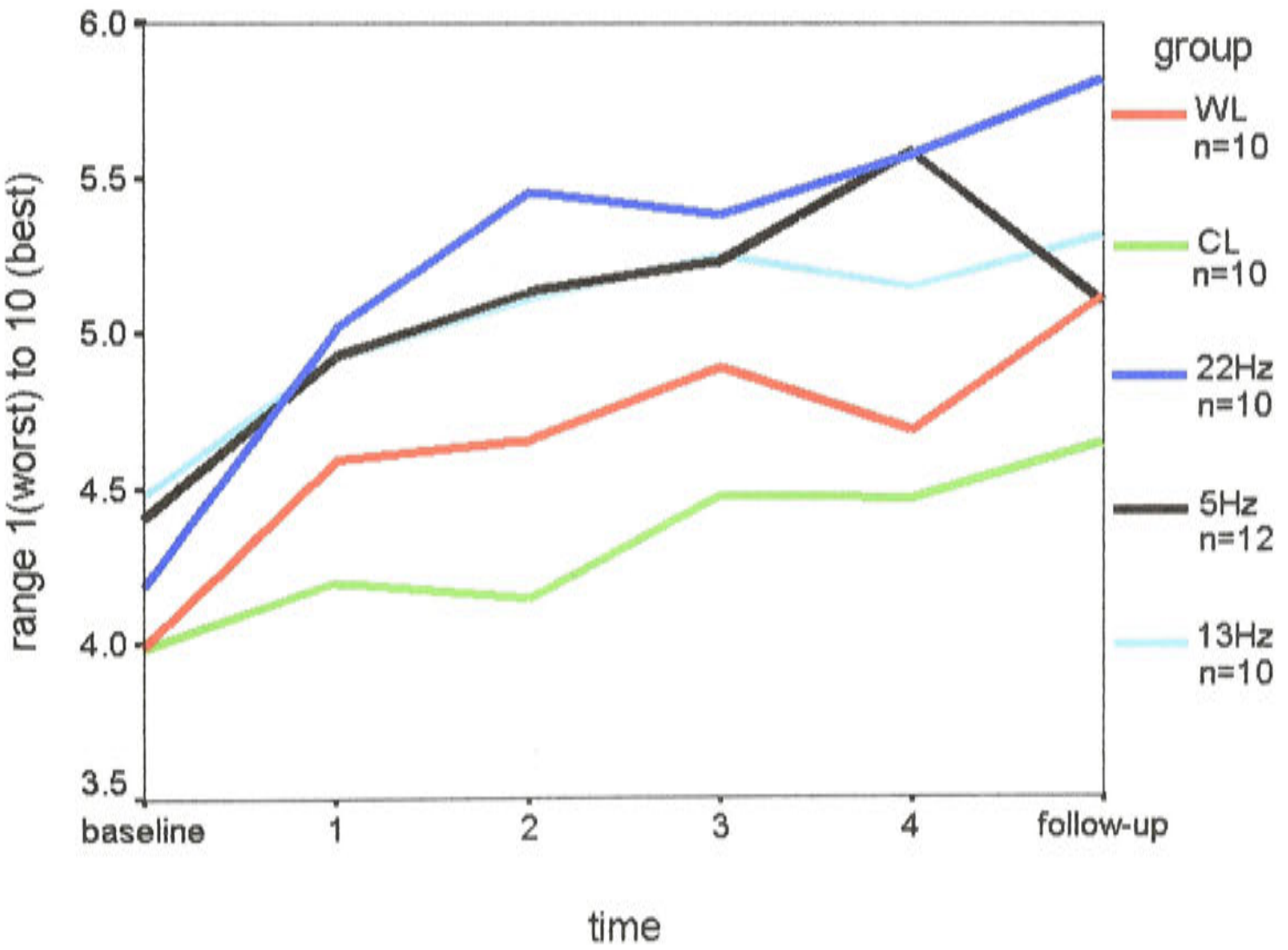



Figure 4.20 Mean waking mood across six weeks of sleep diary.

**4.10.11 Sleep, EEG and mood inter-relationships:** Correlations between sleep variables and EEG were small to moderate across sessions (see Appendix L, for correlation matrix). Sleep onset latency failed to correlate with global theta r = -.08 to -.01, p > .05; global alpha, r =-.15 to -.02, p > .05; or global beta, r =-.09 to .13, p < .05. Similarly, there was little to no relationship between sleep efficiency and global EEG, r = -.07 to .07, p > .05, for global theta; r = -.13 to -.01, p > .05, for global alpha; and r = -.20 to -.06, p < .05, for global beta. Number of 'night wakings', however, was positively correlated with global EEG measures, r = .12 to .26, p > .05, for global theta; r = .16, p > .05, to .31, p < .05, for global alpha; and r = .15, p > .05, to .30, p < .05, for global beta, indicating that number of night wakings was associated with increased EEG amplitude in the alpha and beta bandwidths.

Table: 4.3 Ranges for Pearson's Correlation Coefficients for frontal, midline and posterior broadband EEG and sleep variables across sessions.

| | Sleep onset | Sleep efficiency | Night wakings |
|---|---|---|---|
| 1. sleep onset | 1.0 | | |
| 2. sleep efficiency | [-.85**,-.78**] | 1.0 | |
| 3. night wakings | [-.21, -.05] | [-.10,.06] | 1.0 |
| 4. frontal theta | [-.05,.06] | [-.10,.09] | [.03,.18] |
| 5. frontal alpha | [-.11,.03] | [-.15,.01] | [.11,.26*] |
| 6. frontal beta | [-.05, .14] | [-.19, -.08] | [-.34**,.20] |
| 7. midline theta | [-.08, .01] | [-.09, .06] | [.10, .43**] |
| 8. midline alpha | [-.13, .10] | [-.17, -.02] | [.10, .26*] |
| 9. midline beta | [-.10, .13] | [-.19, -.04] | [.10, .28*] |
| 10. posterior theta | [-.15, -.08] | [-.06, .07] | [.15, .34**] |
| 11. posterior alpha | [-.17, .25] | [-.06, .02] | [.22, .37**] |
| 12. posterior beta | [-.08, .12] | [-.20, -.05] | [.17, .34**] |

*significance LE .05 **significance LE .01

On closer inspection, see Table 4.3, it was found that number of night wakings tended to correlate more strongly with mean posterior broadband EEG than with frontal or midline broadband EEG, suggesting that participants reported more night

wakings with increased posterior theta, alpha and beta amplitudes than with increased frontal or midline EEG amplitude. For example, at baseline, night wakings were significantly correlated with posterior theta, $r = .33$, $p < .05$, but not with frontal, $r = .12$, $p > .05$, or midline theta, $r = .27$, $p > .05$ with a similar pattern observed across sessions.

Sleep variables showed only small to moderate correlations with psychological variables across sessions. Depression was associated with increased sleep onset with positive correlations between sleep onset latency and depression level at baseline, $r = .25$, $p > .05$, session 3, $r = .32$, $p < .05$, and follow-up, $r = .22$, $p > .05$, but not at session 2, $r = -.10$, $p > .05$. Thus, overall, the expected trend of disrupted sleep onset with increasing depression levels was found. Congruent with this finding, sleep onset latency correlated negatively with positive affect at session 2, $r = -.31$, $p < .05$; session 3, $r = -.29$, $p < .05$; and somewhat at follow-up, $r = -.16$, $p > .05$, but not at baseline, $r = .02$, $p > .05$.

Contrary to expectations, sleep onset was unrelated to anxiety levels, $r = .13$ to $.17$, $p > .05$, across sessions. Interestingly, sleep onset was related to photic stimulation response, at session 2, $r = .34$, $p < .01$, but not at other sessions and with alpha asymmetry at session 3, $r = .29$, $p < .05$, but not at other sessions. Thus sporadically, enhanced photic driving responses and increased alpha asymmetry were associated with disrupted sleep onset.

Sleep efficiency was generally unrelated to depression with small non-significant correlations across sessions, $r = -21$ to $.16$, $p > .05$. Likewise for anxiety, small non-

significant inverse correlations were found across sessions, $r = -.11$ to $-.07$, $p > .05$. Sleep efficiency correlated with positive affect during the active photic stimulation period only, $r = .26$, $p < .05$, at session 2, and $r = .29$, $p < .05$, at session 3, with small non-significant positive correlations at baseline and follow-up. However, there was no relationship between sleep efficiency and negative affect, $r = -.12$ to $.13$, $p > 0.5$, across sessions. A consistent negative correlation was found between sleep efficiency and photic stimulation response and alpha asymmetry across sessions, with a significant correlation with photic stimulation response, r -.30, $p < .05$, and alpha asymmetry, $r = -.29$, $p < .05$, at session 3, but not at other sessions.

Contrary to expectations, depression was associated with a lower number of night wakings at baseline as evidenced by a significant inverse correlation, $r = -.28$, $p < .05$. This pattern was not consistent, with near zero correlations revealed at other sessions. Similarly, number of night wakings was inversely related to anxiety levels at baseline, $r = -.34$, $p < .05$, but a near zero correlation at follow-up. Night wakings were unrelated to positive affect, $r = .04$ to $.22$, $p > .05$, and negative affect, $r = -.11$ to $.15$, $p > .05$, across all sessions.

**4.10.12 Menstrual cycle and EEG**: Week of menstrual cycle was ascertained at baseline because of the unusual positive correlation between week of menstrual cycle and positive affect in Study 1, suggesting increased positive affect as the luteal phase of the menstrual cycle progressed. The positive correlation between positive affect and week of menstrual cycle was not replicated in the current study, $r = .08$, $p > .05$. Week of menstrual cycle was also unrelated to negative affect, $r = -.01$, $p > .05$, depression, $r = -.17$, $p > .05$, and anxiety, $r = -.17$, $p > .05$. Unlike Study 1, week of

menstrual cycle was also unrelated to EEG, $r = .10$, $p > .05$, for global theta; $r = .28$, $p > .05$, for global alpha, and, $r = .17$, $p > .05$, for global beta.

### 4.11 Discussion

The main aim of this study was to assess the therapeutic utility of photic stimulation in the treatment of mood and sleep disturbance. As outlined in Chapter 3, depression can be viewed as a stress-related disorder characterised by over-stimulation of arousal systems and dysregulated feedback mechanisms, with dire consequences for the regulation of mood and sleep (Chrousos & Gold, 1992). Based on the premise that frequent elicitation of relaxation responses 're-trains' over-taxed arousal systems and helps to restore homeostasis (Benson, 1975, 1985), this study sought to test the ability of photic stimulation to decrease arousal and ameliorate dysphoria and sleep disturbance in a clinically depressed group.

This study attempted to mimic the therapeutic setting where the therapist teaches relaxation skills and encourages regular home practice, in order to facilitate training effects which generalise beyond the practice period, because of frequent elicitation of relaxation responses, which then become over-learned and almost automatic (Agras et al., 1980; Everly & Benson, 1989). This was achieved by, providing participants with light masks, to take home and use on a daily basis, for a period of one month. It was anticipated that with practice, over-stimulated arousal systems would adapt and become less reactive to threat and challenge. As arousal systems normalised, it was hypothesised that mood, depression, anxiety, and sleep would improve over the course of the study, with concomitant changes in EEG functioning and decreased responsivity to light flicker.

**4.11.1 EEG findings:** This study supported Henriques and Davidson's (1991) robust finding of alpha asymmetry in depression. Participants showed greater left midfrontal alpha relative to right midfrontal alpha, indicating left frontal cortical deactivation relative to the right. Alpha asymmetry was found to be relatively stable over time despite improvements in mood. This was also reflected in the finding that alpha asymmetry was unrelated to depression level, with small, non-significant positive correlations found. Increasing alpha asymmetry, however, was associated with negative affect with significant positive correlations. Given that alpha asymmetry was due to an increase in left midfrontal alpha in the current study, this finding supports Davidson's proposal that deactivation of the left frontal cortex diminishes the ability to experience positive emotions and results in a preponderance of negative emotions. Alpha asymmetry, therefore, is a trait marker for depression which influences affective style and increases the risk of subsequent depressive episodes (Davidson, 1998b, 2001; Tomarken & Keener, 1998). In previous work, Davidson and colleagues found that depressives had increased left frontal alpha in comparison to controls, but right frontal alpha was within normal limits. In the current study this was not supported, with both left and right alpha magnitude approximately 1.5 standard deviations below the norm at baseline.

Another characteristic of the current sample was the finding of low frontal theta and beta, with approximately 45 to 50% of participants showing low frontal theta, and 30-35% displaying low frontal beta, two standard deviations or more below the normative database. Previous research has also found low frontal theta in depressed subjects accompanied by high frontal and posterior beta (Ohashi, 1994). Ohashi's

findings are congruent with the arousal model of depression as high beta indicates cortical arousal and is associated with anxiety, and low theta possibly indicating a diminished ability to 'switch off' and relax. In the current sample, however, the finding of low frontal theta was not accompanied by high beta. The findings in this study only partially support the hyper arousal hypothesis of depression, with low frontal theta possibly reflecting difficulties initiating relaxation responses that in turn prolong sleep onset. It is possible that the sample used in the current study were not as 'stressed' or hyperaroused as that used by Ohashi, which would account for the absence of elevated beta. The presence of low frontal theta, however, is possibly a characteristic of the depressed and sleep disturbed sample used in this study, as suggested by Ohashi (1994).

**4.11.2 Brainwave entrainment**: The current study showed strong evidence for photic driving in response to 10Hz photic stimulation with 72% of participants displaying increased amplitude in the 10Hz bandwidth and recruitment of the response across the cortex into frontal areas. Of those who did respond, 79% demonstrated a 50% or more increase in cortical alpha in response to photic stimulation. Given that strong entrainment responses were not found with 5Hz, 13Hz or 22Hz AVS in Study 1, this supports the claim that entrainment effects are more likely to be found at frequencies close to occipital peak alpha frequencies (Niedermeyer, 1997; Silberstein, 1995b; Toman, 1941).

In addition to entrainment responses many participants also showed clear harmonic responses. Forty two percent of participants showed a 50% or more increase in the 20Hz bandwidth in response to 10Hz photic stimulation in occipital leads, and in

some people also in central and frontal leads (see Figure 4.7b). This is consistent with other research which showed harmonic effects in beta bandwidths in response to 10Hz photic stimulation (Rosenfeld et al., 1997). Larger harmonic responses are more likely to be found when using photic frequencies close to the individual's peak alpha frequency (Lazarev et al., 2001), but have also been noted with 5Hz and 20Hz photic stimulation (Kikuchi et al., 2002).

The presence of harmonic responses with photic stimulation complicates the use brainwave entrainment tools for therapeutic purposes. Entrainment at a particular frequency may be producing cortical changes at harmonics of the first, second, third, fourth and possibly higher orders (Kikuchi et al., 2002; Rosenfeld et al., 1997). For example, using a light flicker of 10Hz, with the intention of enhancing alpha rhythms and eliciting concomitant relaxation effects, may be offset with enhancement of beta rhythms within the 20Hz, 30Hz or even 40Hz bandwidths caused by harmonic responses which can be accompanied by increased alertness, agitation, or even anxiety (Walter, 1953; Walter & Walter, 1949). Prior to using brainwave entrainment tools, it may be necessary to assess individual responsivity to the driving stimulus and the extent of harmonic effects.

While there was strong evidence for entrainment to 10Hz photic stimulation, entrainment responses to experimental frequencies were not directly assessed. Rather, evidence of entrainment was sought by assessing if regular daily light mask use produced 'carry-over effects' evidenced by increases in EEG magnitude at entrainment frequencies in the eyes closed EEG. There was little evidence of entrainment 'carry-over' effects in the data, with very small non-significant effects

revealed. Possible EEG training effects were present in the 5Hz group who showed a sharp increase in EEG magnitude in the 5Hz bandwidth after 2 weeks of light therapy. This effect did not persist, however, with a return to baseline levels by the end of 4 weeks of light therapy. All remaining groups, 13Hz, 22Hz, CL, and including the WL group, who received no light therapy, also showed very small non-significant increases in the 5Hz bandwidth across sessions. It is possible that participants in the photic stimulation groups showed increases in 5Hz EEG magnitude because of harmonic responses. Given that the control group also displayed increases in 5Hz EEG magnitude, however, this is not likely.

The most likely explanation for the small increase in 5Hz EEG activity seen across groups is the presence of general relaxation responses or an increase in sleepiness both of which are associated with increased theta activity (Banquet, 1973; Corby et al., 1978; Dierks et al., 1989; Jacobs et al., 1996; Jacobs & Lubar, 1989; Saxby & Penniston, 1995; Strijkstra et al., 2003). Over the course of time as participants became familiarised with the EEG procedure it is quite likely they exhibited greater relaxation responses during recording sessions. This is further supported by reports by some participants of increased fatigue during the recording session, despite efforts to ensure that participants did not fall asleep during EEG recordings. Given that theta is associated with cortical deactivation and drowsiness this would also account for these small increases in theta EEG (Fried, 1993; Jacobs & Lubar, 1989; Sterman, 1996). To assess if Lightmask frequencies produced photic driving responses in this study it would be necessary to record changes in EEG magnitude while participants were exposed to their Lightmask frequency, however, in order to maintain the double blind condition, this was not done.

To address this issue, at the end the study, frequency following responses to 5Hz, 13Hz and 22Hz photic stimulation were assessed in a 15year old unmedicated female with mild pathology (SCL-90-R depression T-score=71, anxiety T-score=67, and general severity index T-score=71) and responsive to 10Hz photic stimulation, in order to ascertain if entrainment to 10Hz photic stimulation was also accompanied by entrainment at other frequencies, or limited to frequencies close to the peak alpha frequency. Using the same procedures and equipment as described above, photic stimulation of 5Hz, 10Hz, 13Hz, and 22Hz was delivered using the Mind-Gear light and sound machine and EEG recorded for three minutes.

As can be seen in Figure 4.21, the client had a dominant alpha frequency of approximately 10Hz. Figure 4.22 shows clear entrainment responses to 10Hz photic stimulation with an increase in EEG amplitude in the 10Hz bandwidth and a clear harmonic response noted at 20Hz both occipitally and extending into frontal regions. Photic stimulation at 5Hz (Figure 4.23) shows no entrainment response in the 4-6Hz bandwidth, but marked harmonic responses can be seen in the 10Hz and 20Hz bandwidths. Figure 4.24 shows clear frequency following to 13Hz photic stimulation with entrainment evident in the 12-14Hz bandwidth. Interestingly, photic stimulation at 13Hz resulted in attenuation of EEG amplitude at the sub-harmonic level of 6.5Hz. No first order entrainment effects were seen with photic stimulation at 22Hz with little to no increase in EEG amplitude in the 22Hz bandwidth. Sub-harmonic responses were noted, however, with a small increase in EEG amplitude within the 9-11Hz bandwidth in occipital leads only.

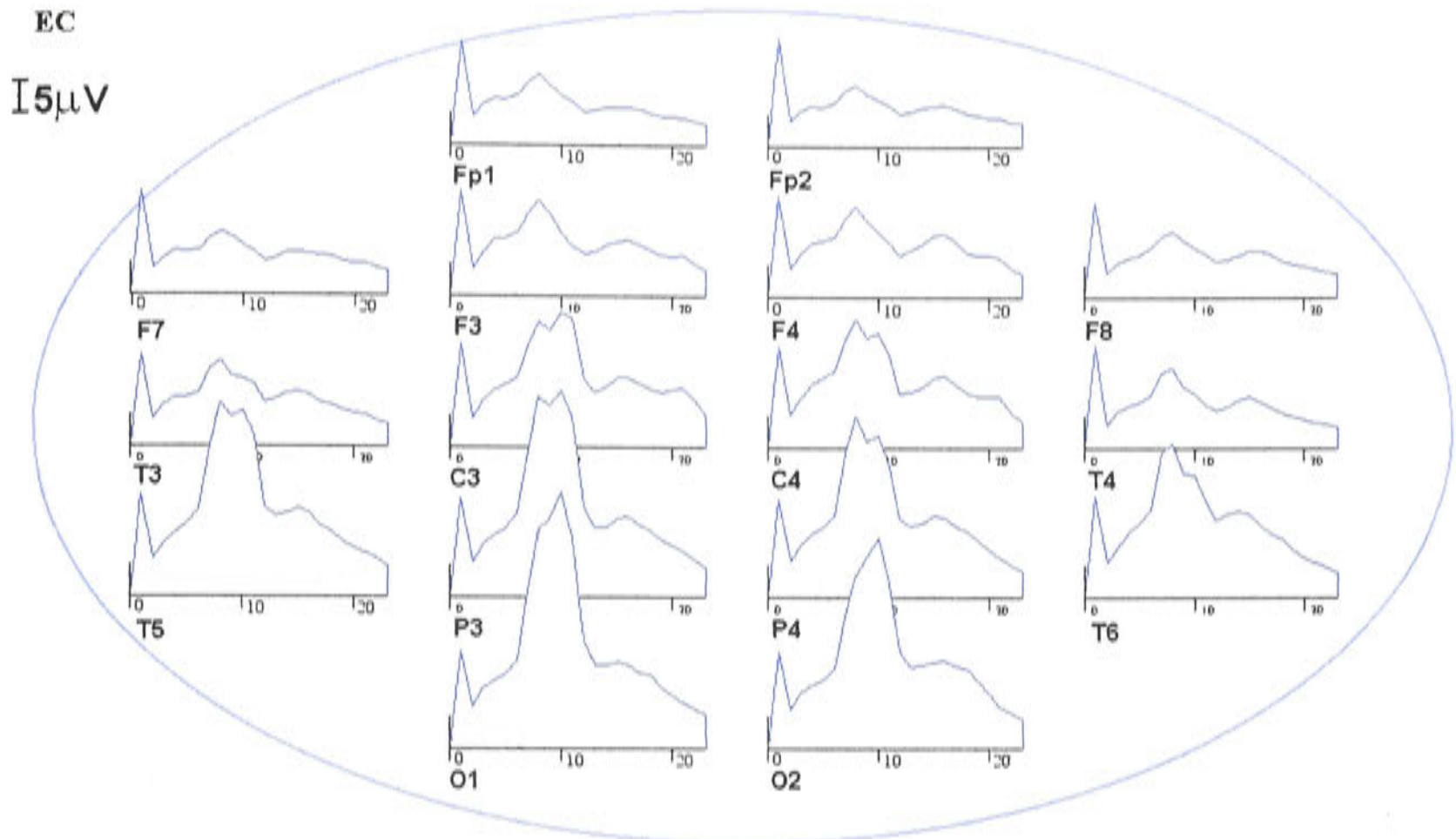



Figure 4.21 Spectral plot of 'eyes closed' EEG across 16 scalp sites (0-23Hz bandwidth with markers at 0, 10 and 20Hz) showing a 10Hz dominant alpha frequency in a 15year old, mild to moderately depressed female, with anxiety symptoms.

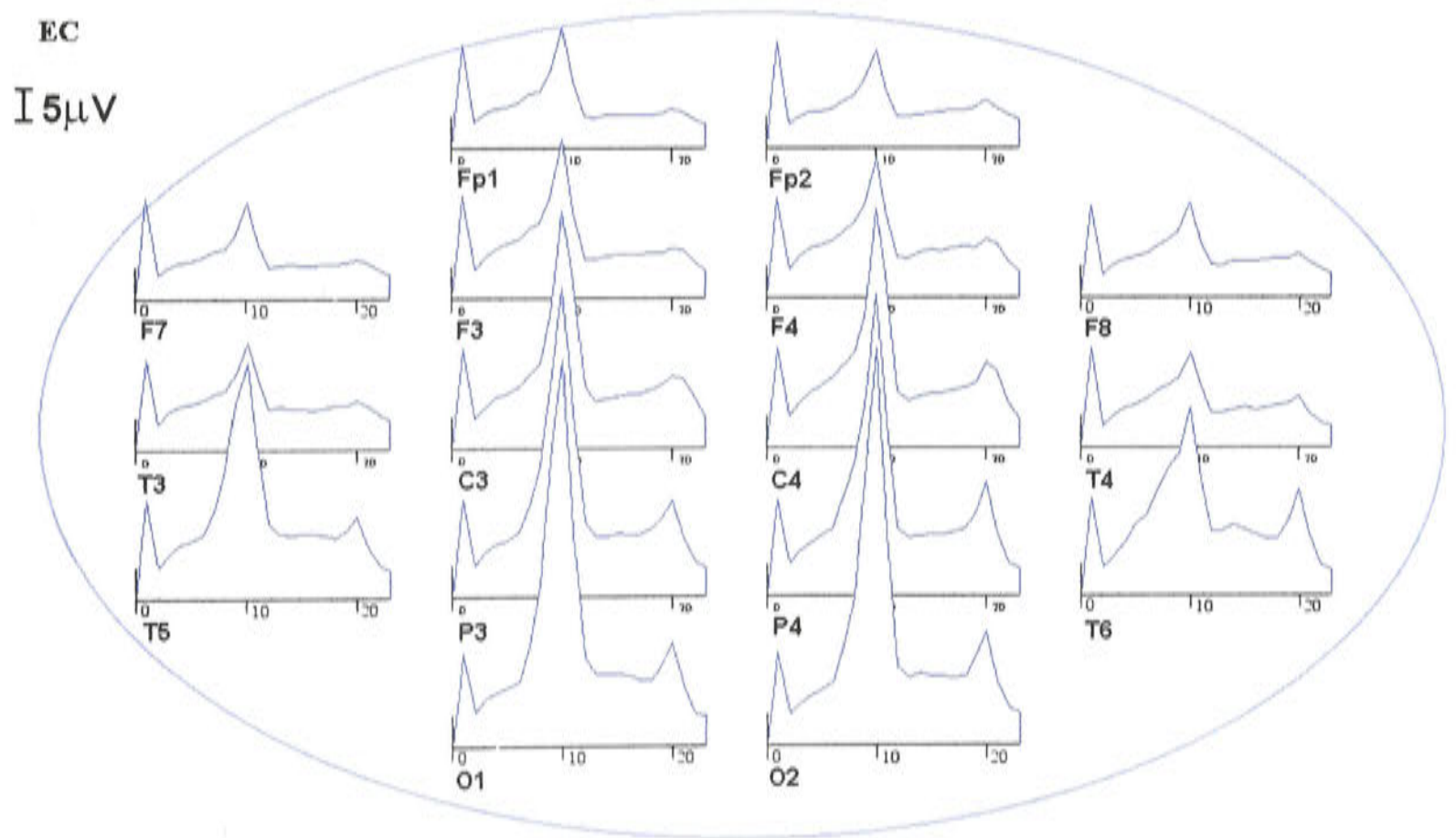



Figure 4.22 Response to 10Hz photic stimulation in the same 15yr old female showing a notable increase in 10Hz dominant alpha frequency and clear 20Hz harmonic effects extending across the cortex.

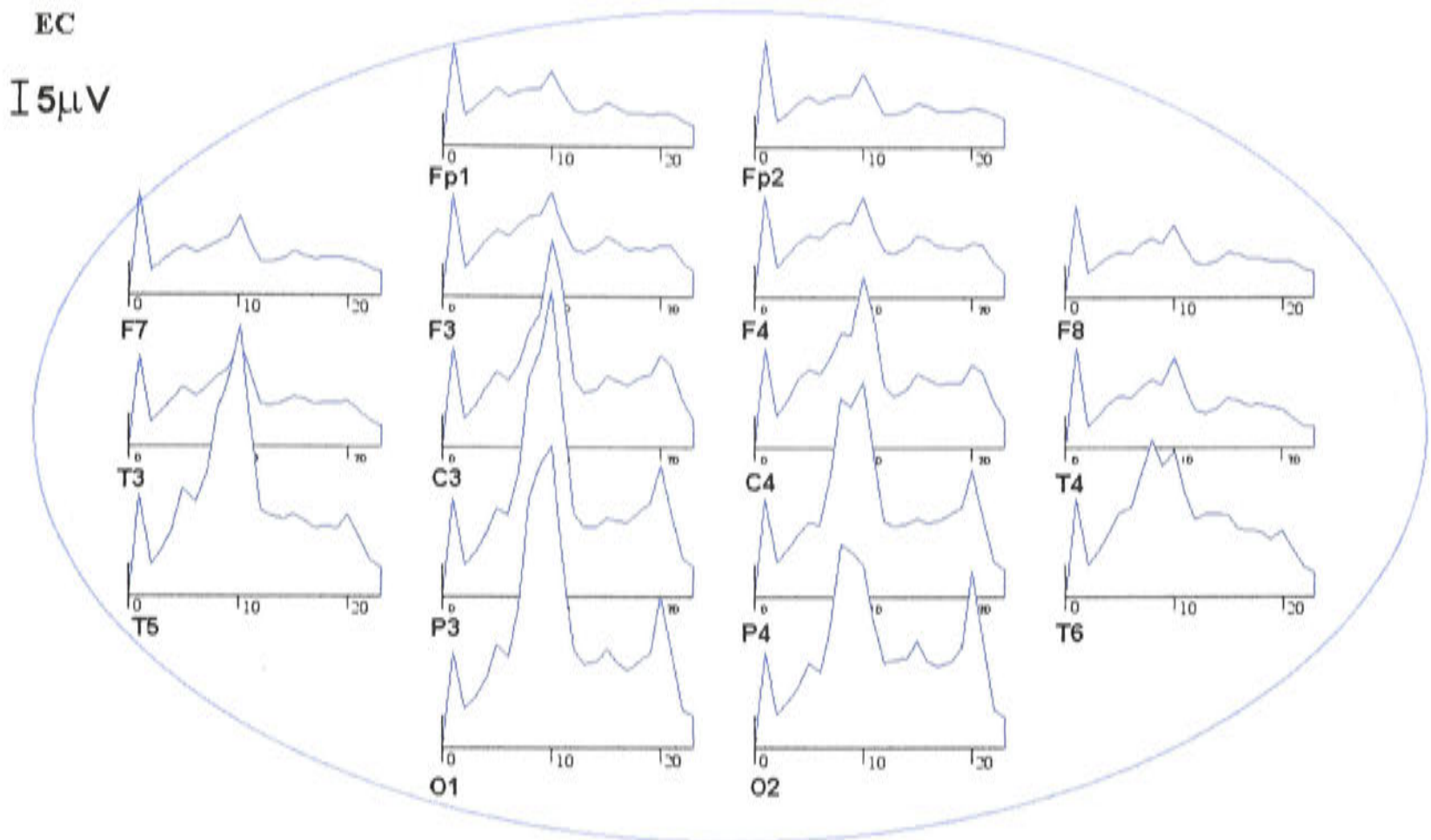



Figure 4.23 Response to 5Hz photic stimulation in the same 15yr old female showing little evidence of entrainment at 5Hz, but harmonics visible at 10Hz and 20Hz.

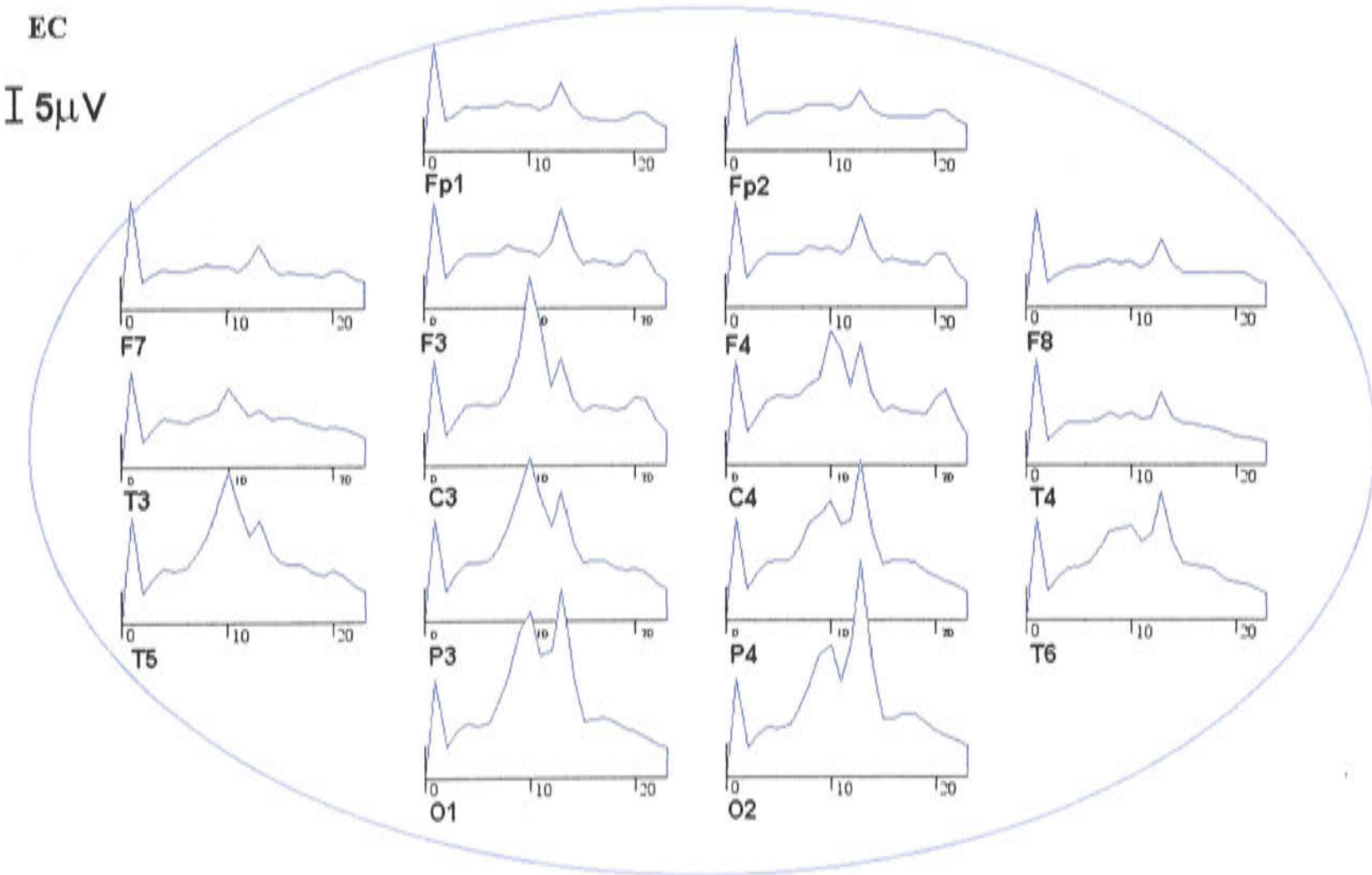



Figure 4.24 Response to 13hz photic stimulation in the same 15yr old female showing entrainment in the 13-14Hz bandwidth, a decrease in dominant 10Hz alpha, and a decrease in 6.5Hz.

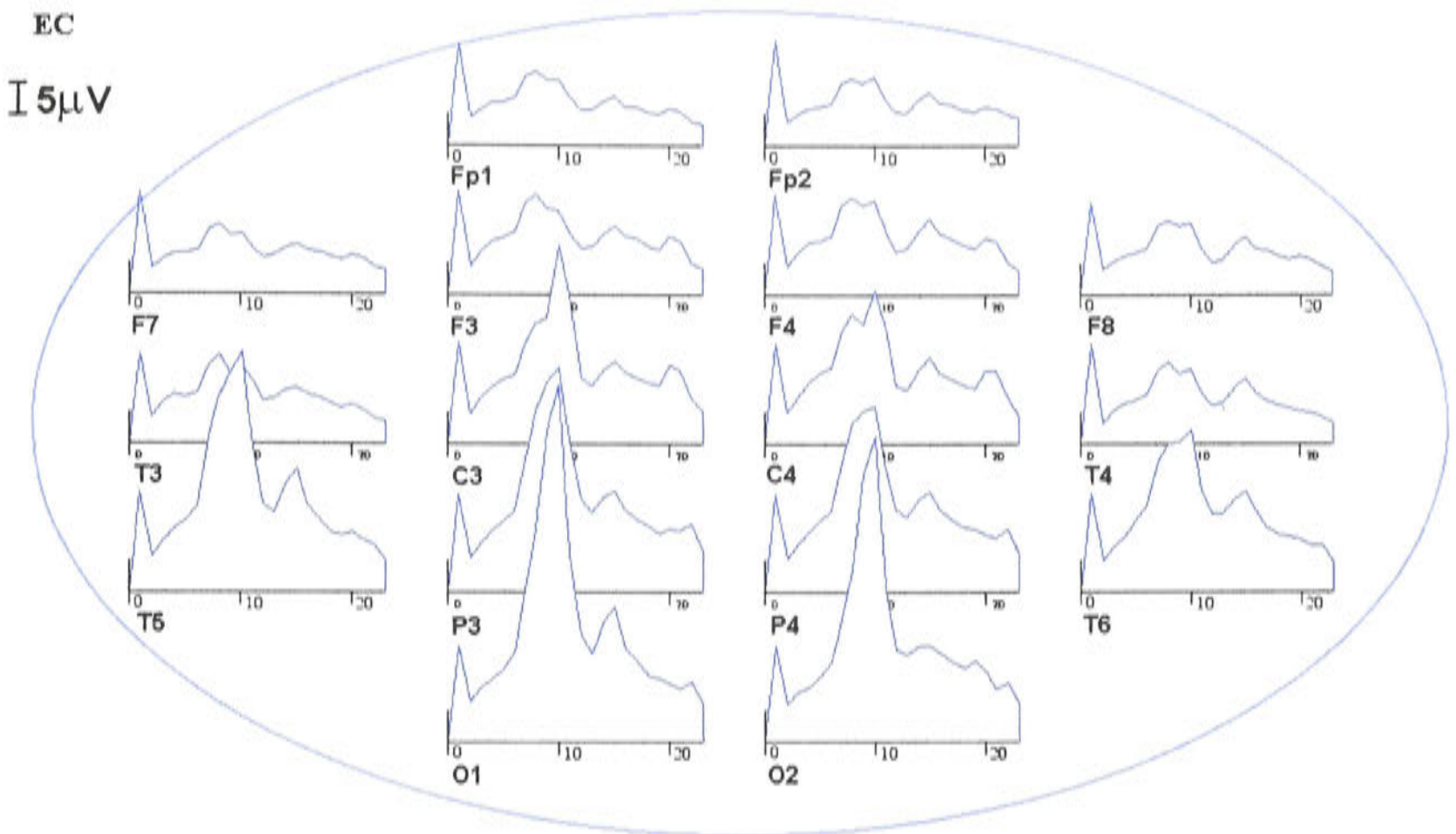



Figure 4.25 Response to 22Hz photic stimulation in the same 15 yr old female showing no entrainment in the 22Hz band, but a sub-harmonic response at 11Hz visible in occipital leads.

Therefore, in a sample of one it was shown that photic driving responses occur not only at the dominant alpha frequency, but also at entrainment frequencies quite distant from the peak alpha rhythm, and with evidence of harmonic responses at frequencies distal from the peak alpha frequency. This is consistent with previous research which shows photic driving across a wide range of frequencies with recruitment of harmonic responses across the cortex (Mundy-Castle, 1953; Wada et al., 1994; Walter & Walter, 1949). Thus it is plausible in the current study that participants who demonstrated photic driving to 10Hz stimulation, were also experiencing frequency following when using their light mask at home. Evidence for entrainment 'carry-over' effects following regular daily light mask use were not found when assessing eyes closed EEG, therefore showing that while entrainment

effects may have been occurring during light mask use at home, they did not have an enduring influence on the EEG.

Some caution needs to be exercised before generalising these findings. The data were collected from a 15year old mild to moderately depressed client with reported sleep disturbance. The age difference between this client and the sample used in the current study may prohibit direct comparisons as photic driving is higher in children and adolescents than in adults (Takahashi, 1993). In addition, while entrainment responses were found using the 'Mind Gear' light and sound machine in one client, it is possible that photic driving responses using the Lightmask may not be equivalent. This needs to be addressed in future research.

Failure to find clear evidence of entrainment to Lightmask frequencies in the current study, may have been contributed to by harmonic effects which could have masked intended EEG changes at entrainment frequencies by increasing power in opposing EEG frequencies. Consequently, mood and relaxation effects usually associated with particular cortical EEG frequencies, may have been offset by enhancing EEG frequencies and mood effects antithetical to the desired outcome (Rosenfeld et al., 1997).

Evidence for the effectiveness of photic stimulation to produce immediate relaxation responses, however, can be found in participants' rated subjective well-being immediately after light mask use. Those receiving 5Hz, 13Hz, or 22Hz photic stimulation tended to report higher well-being than participants exposed to continuous light, but this effect was small and not significant. The most plausible

explanation for this small effect is the greater relaxation responses elicited by active photic stimulation in comparison to continuous light. Continuous light lacks one of Benson's main components for producing relaxation responses, 'a repetitive stimulus on which to focus'. It is plausible that participants habituated more readily to continuous light than to perceptible light flicker, which may hold attention and suspend mental activity for longer with greater opportunity to elicit relaxation responses before habituation occurs. These findings are consistent with previous research which showed that photic stimulation produces immediate relaxation effects, but does not have enduring long term effects on psychological state (Ossebaard, 2000).

**4.11.3 Mood and sleep:** Mood improved significantly across sessions for all groups with strong placebo and non-specific effects contributing to results. In the current study it was anticipated that improvements in mood would be evident firstly because of the influence of expectancy effects, which were manipulated to produce positive expectancies, and secondly, as a function of the different photic stimulation frequencies used. Positive affect, negative affect, depression, anxiety and symptom severity all changed in predicted directions, but largely independently of the photic stimulation frequencies used. Specifically, positive affect increased for all groups over the course of the study, including the waitlist group who received no intervention. Correspondingly, negative affect decreased for all groups over the study period. The observed effects of increased positive affect and decreased negative affect with 13Hz stimulation over and above the other frequencies of stimulation, which was found in Study 1, was not repeated. Similarly, the hypothesised greater increases in positive affect and decreases in negative affect,

attributable to active photic stimulation in comparison to WL and CL, was not found as all groups performed in a similar manner. In addition, it was anticipated that depression and symptom severity would decrease over the course of the study for all groups, with greater improvement in participants using active photic stimulation. While all groups showed improvement in depression and symptom severity over the study period, photic stimulation contributed little to these effects.

Likewise, for anxiety, all groups showed significant decreases in anxiety from baseline to follow-up. It was anticipated that 5Hz photic stimulation would benefit high anxiety participants, while 22Hz photic stimulation would benefit those with low anxiety. In the current study, however, different frequencies of photic stimulation did not preferentially influence high or low anxiety levels as predicted. Therefore entrainment effects were not responsible for the decrease in anxiety levels observed, rather, relaxation responses coupled with strong placebo and non-specific effects appear to have played a significant role.

These findings are consistent with previous research which found placebo to be as effective as active treatment using mind-machines in its ability to induce relaxation responses (Walach, 1998). In Walach's study, the use of a sophisticated mind machine may have been sufficient to elicit strong placebo responses from users, despite no active treatment. Similarly in the current study, the use of the light mask may have induced placebo effects. Contrary to this, however, is the finding that even the waitlist group improved over time with similar enhancements in mood and decreases in depression and anxiety as the active and placebo groups. This suggests,

therefore, that while light mask use may have elicited beneficial placebo effects and possibly some subtle relaxation effects, other non-specific effects were also present.

A similar picture emerged for sleep. Sleep improved over the course of the study with a decrease in number of night wakings, improved sleep efficiency and waking mood, but no change in sleep onset latency for all groups. These changes however, were largely independent of group membership, with those in the WL and CL groups reporting improvements in sleep similar to the active photic stimulation groups.

There were some patterns of response with regard to sleep that could potentially be related to entrainment effects. For example, 5Hz photic stimulation decreased sleep onset latency from 46 minutes at baseline, to below 30 minutes for the entire intervention phase, which is under that recommended for clinical significance (Epsie, 2002), with a return to baseline measures by follow-up. Unexpectedly, CL also reduced time to sleep onset to below clinical levels after one week of use with a persistence of the effect to follow-up. It is possible that the latter response was due to entrainment effects caused by sub-harmonic responses to 64hz stimulation that impacted on the EEG at lower frequencies and, in turn, reduced sleep onset in a manner similar to 5Hz stimulation. This assertion is difficult to sustain, however, with the finding that waking mood was higher in the active photic stimulation groups in comparison to WL or CL, possibly due to photic driving responses influencing aspects of sleep quality that were not directly assessed in this study, but that nevertheless impacted on waking mood. The finding of a marked decrease in sleep onset latency with 5Hz photic stimulation with little to no change afforded by 13Hz or 22Hz stimulation further supports the view that frequency following may be

involved, as increasing theta activity (5Hz photic stimulation) is conducive to sleep initiation.

An alternative explanation, however, is that both CL and active photic stimulation produce relaxation and entrainment effects, with active photic stimulation producing greater relaxation effects than CL which consequently impacts to a greater degree on waking mood. Entrainment initiated by 13Hz or 22Hz photic stimulation would produce relaxation effects as demonstrated in Study 1, but would also act to increase cortical arousal, and drive the cortex away from slow theta oscillations which are necessary for sleep initiation; and therefore work in opposition to relaxation effects and have little impact on sleep onset. While these conclusions offer some support for the presence of relaxation and entrainment effects with photic stimulation, they were drawn from non-significant trends, based on inferences within the data and without direct evidence of entrainment with Lightmask use.

Confounding the interpretation of results is the finding that even the WL group demonstrated improvement in mood, depression, anxiety and sleep similar to that seen in the other groups. This points to the presence of other non-specific effects. For example, all participants visited a psychologist in the psychology department at the local hospital four times over the course of 3 months, with at least 6 hours of contact time with a professional. Since 48% of the sample reported, at initial interview, that they were currently seeing a health professional for their depression, it can not be ruled out that the building of a therapeutic alliance with a health professional over the course of the study may have contributed to the improvements noted (Ilardi & Craighead, 1994; Mayberg et al., 2002). Further to this point, there

was no difference between groups for professional help seeking behaviour, therefore the effect observed in the wait-list group cannot be attributed to initial lower health professional contact which may have been ameliorated by study participation.

In addition, all participants were required to maintain sleep diaries for six weeks over a 3 month period, and completed mood questionnaires four times over this period, thus highlighting to participants the extent of their sleep and mood problems. Some participants reported that merely noting sleep patterns and becoming aware of their daily mood, was sufficient to motivate behaviour change and improve lifestyle factors related to mood and sleep hygiene. These ad hoc reports suggest that diary keeping was sufficient to motivate some people to change aspects of depressive behaviour which consequently impacted on mood and sleep. Within a stress and coping model, diary keeping can be viewed as a problem focussed coping strategy providing participants with a course of action which enables them to better cope and adapt with the stress of depression (Lazarus & Folkman, 1984). Successful adaptation also increases self-efficacy and enhances positive expectancies which foster hope that 'things will get better' (Bandura & Adams, 1977; Lazarus, 1991a). Requiring participants to attend a health setting for the experiment can also elicit positive expectancies and generate hope that help is being sought and recovery will ensue (Frank, 1973, 1982b; Ilardi & Craighead, 1994). Expectancy and placebo effects are well noted in clinical research and have been shown to induce therapeutic effects in psychological and physiological parameters and even produce responses similar to those induced by active pharmacological treatment (Leuchter, Cook, Witte, Morgan, & Abrams, 2002; Mayberg et al., 2002; Schatzberg & Kraemer, 2000).

### 4.11.4 Photic stimulation, EEG, mood and sleep inter-relationships

As previously noted, photic driving response to 10Hz photic stimulation was found in a substantial proportion of the sample, but contrary to previous research, it was found to be independent of depression level. Frequency following responses did not decrease over the course of the study as purported by Pockberger (1985). Instead, they behaved in a more stable manner across time possibly reflecting a trait like aspect of depression. Other research has reported higher frequency following responses with alpha stimulation in individuals with low alpha power and attenuated responses in those with high alpha power (Rosenfeld et al., 1997). This is somewhat congruent with early work by Shagass (1955) who found high anxiety, depressed females, possibly with low alpha power, had higher photic driving responses in comparison to low anxiety depressed females, most likely with higher alpha power. Similarly, Kumano (1997) found that physiologically and emotionally reactive subjects, indicating heightened arousal, also showed higher photic driving responses in comparison to more stable subjects. In the current study, however, there was no correlation between heightened arousal, as indicated by anxiety, and photic stimulation response, nor photic stimulation and alpha power (see Appendix L). In contrast, however, photic stimulation was inversely related to sleep and subjective relaxation. Those with higher photic stimulation responsivity reported feeling less relaxed, had higher sleep onset latencies, and poorer sleep efficiency suggesting that some form of physiological arousal was involved and related to photic driving responsivity as indicated by previous research.

Previous research has found a relationship between anxiety and psychological distress and increased cortical activity such as beta rhythms which reflect increased

arousal (Matousek, 1991; Nofzinger et al., 2000; Nystrom et al., 1986). In the current study, however, increasing pathology, such as depression, anxiety and symptom severity tended to be associated with a decrease in amplitude across all bandwidths, with a decrease in theta, alpha and beta amplitude for depression and symptom severity, and attenuated alpha and beta amplitude for anxiety, as evidenced by small non-significant correlations. This decrease in EEG amplitude with increasing pathology reflects the findings of Nandrino (1994) who found that depressive individuals have less complexity in their EEG in comparison to their never depressed counterparts, with increasing stability or loss of variation with increasing number of depressive episodes. Nandrino argues that a healthy cortical system is chaotic and complex in nature and therefore able to adapt when required, whereas the increased stability found in the EEG of depressives reflects underlying pathology and is associated with maladaptive behaviours such as ruminative thinking and psychomotor retardation (Nandrino et al., 1994; Pezard & Nandrino, 2001).

Significant correlations between depression and sleep were found. Increasing severity of depression was associated with delays in sleep onset as expected. Unexpectedly, the number of night wakings decreased as depression deteriorated, yet sleep efficiency remained compromised as evidenced by small inverse correlations between sleep efficiency and depression. Therefore this small decline in sleep efficiency was not due to an increase in nigh time waking, but rather related to difficulties with sleep onset or early morning waking. These findings are consistent with the hyper-arousal hypothesis of depression wherein increased physiological and cognitive arousal leads to disrupted sleep wake cycles and delays in sleep onset. A further symptom of dysregulated arousal in depression is the advance in the timing of

nocturnal cortisol secretion that leads to early morning waking and difficulties returning to sleep after waking, contributing to sleep loss and further compromising sleep efficiency (Wallace, 1996). In the current study, a significant positive correlation was found between night time waking and global alpha and beta. This adds further support to the hypothesis that arousal mechanisms play a role in sleep disruption in depressed individuals, as those with higher alpha and beta amplitude reported increased night time waking, which contributes to disruption of sleep continuity.

Study 1 revealed the unexpected finding of increased positive affect as menstrual cycle progressed. In the current study, however, menstrual cycle was unrelated to positive affect and all experimental variables. This finding is not surprising given the small number of subjects used to calculate these statistics (n=23), coupled with a limited range of variability due to the use of a depressed sample. A small, almost significant positive correlation was found between menstrual cycle and alpha, indicating an increase in alpha activity, or increased cortical deactivation as the luteal phase of the menstrual cycle approached.

### 4.12 Limitations of the study

A limitation of the current study was the inability to assess if the CL group, receiving 64Hz continuous light, were in fact a placebo control and unaffected by light flicker, or conversely, experiencing photic driving induced by lower order harmonics at 32Hz, 16Hz, or even 8Hz stimulation. While 64Hz is beyond the flicker fusion point, some studies report EEG responses to higher frequencies caused by sub-harmonics (Takahashi, 1993). For example, some light sensitive epileptics are much

more likely to show paroxysmal EEG spikes to a 50Hz video monitor than a 100Hz video monitor because of the 25Hz harmonic produced by the 50Hz monitor (Badinand-Hubert et al., 1998; Harding, Fylan, & Edson, 1997). Fluorescent lighting, which flickers at ~ 50Hz, has also been shown to impact on the EEG by attenuating alpha rhythms and found to increase arousal and decrease performance in some individuals (Kuller & Laike, 1998). Future research needs to assess the impact of lower order harmonics, not only on the EEG, but also on mood and sleep parameters. It is possible in this study that some of the effects associated with CL can be attributed to harmonic effects.

A further limitation of the current study was the inability to directly assess entrainment responses to the take home Lightmasks participants used. As outlined above, in order to maintain the double blind condition, the experimenter doing the EEG was not able to assess frequency following responses to the Lightmask without risking exposing the frequency allocations. To my knowledge there is no published data assessing photic driving responses in the EEG in response to Lightmask stimulation. Previous research, however, has shown that greater entrainment responses are found with red light (Carterette & Symmes, 1952; Takahashi, Tsukahara, & Kaneda, 1981) and can be induced with light intensities as low as 10 Lux (Kumano et al., 1997). Given that in this study, using a sample of one, that clear first order and harmonic photic driving responses were demonstrated in response to experimental frequencies, then it is highly probable that the light masks used in this study exerted entrainment effects. This in turn helps to explain some of the subtle differences that were found in the active photic stimulation groups in comparison to CL or WL.

## 4.13 Conclusions

This study explored the effectiveness of photic stimulation in the treatment of mood and sleep disorder. The results revealed significant decreases in depression, anxiety, and symptom severity accompanied by improvements in sleep efficiency and decreased number of night wakings for all groups over the course of the study. These significant clinical improvements, however, cannot be attributed to re-training of arousal systems induced by frequent elicitation of relaxation responses as hypothesised, as symptom reduction occurred in all groups. Contrary to expectations, the placebo and wait list groups did as well as the active experimental groups with very limited evidence to indicate that photic stimulation contributed to these improvements over and above relaxation responses, positive expectancies and other non-specific effects.

These findings are consistent with many studies which demonstrate the potency of placebo effects to produce changes in emotional and physical processes (Butler & Steptoe, 1986; Leuchter et al., 2002; Mayberg et al., 2002). In addition, placebo responses in clinical studies for depression are quite high, with up to 65-80% of treatment effects obtained with active antidepressant medication able to be reproduced using placebo (Kirsch, 2000). Typically, up to 30-40% of depressed subjects show clinical improvement in response to placebo alone (Brown, Dornseif, & Wernicke, 1988), with placebo capable of maintaining clinical remission for up to 2years (Kirsch, 2000). Other research has shown that effective placebo treatment produces demonstrable changes in brain functioning similar to that obtained with active treatment (Leuchter et al., 2002; Mayberg et al., 2002). The warmth and

enthusiasm of the therapist can also contribute to placebo effects, and has been shown to greatly enhance relaxation responses in study participants (Bench, 2001). Furthermore, stronger placebo responses are usually found in less severely depressed subjects without long term chronicity, not unlike the sample used in the current study (Rush, 2000).

Clinical studies inherently contain many non-specific elements, pertaining to the participant, the experimenter, and the therapeutic setting, which impact on treatment outcomes (Orne, 1962). According to Frank (1973, 1982, 1982b) psychotherapy and clinical research include common features that help to ameliorate the psychological suffering of the client. They provide a therapeutic setting conducive to healing; a supportive therapist equipped with 'expert knowledge'; a very convincing rationale regarding the efficacy of treatment and expected outcomes; and a set procedure through which to operate. Frank asserts, that these factors function to facilitate treatment outcomes by enhancing client self-efficacy, instilling hope for successful treatment, and eliciting positive expectancies, and thereby maximising the impact of placebo responses. This is congruent with the current study, which provided a "healing setting", a trained clinician, a set procedure, a convincing rationale, the manipulation of positive expectancies, and the building of a therapeutic alliance over a three month period.

In the current study, it was anticipated that the various frequencies of stimulation would exert differential effects on mood and sleep that would be evident over and above positive expectancies and placebo responses. This was not found. The only evidence to indicate that photic stimulation may have been more effective than wait-

list or placebo (CL), were some small non-significant trends in predicted directions. In summary, photic stimulation at 5Hz, 13Hz, or 22Hz enhanced well-being and improved waking mood to a small degree over and above exposure to continuous light. Furthermore, 5Hz photic stimulation appeared to reduce arousal, decreasing sleep onset latency, depression and increasing positive affect more than other frequencies to a small degree.

This naturalistic study attempted to reproduce the therapeutic setting by providing participants with a take home light mask in order to maximise treatment effects and enhance compliance. When imitating the 'real life' situation, however, it is difficult to control the interplay of multiple factors that contribute to treatment outcomes, even when controlling for placebo and expectancy effects. There was little evidence in this study of enduring entrainment effects that could be attributed to Lightmask use as hypothesised. Rather, it appears that relaxation effects, coupled with strong positive expectancy effects, and non-specific factors, such as demand characteristics and the development of a therapeutic alliance, contributed to recovery, possibly by increasing self-efficacy and enhancing problem-focused coping (Bandura, 1977; Frank, 1982a; Lazarus, DeLongis, Folkman, & Gruen, 1985). In general, the findings of this study highlight the need for clinicians and researchers to be aware of the powerful impact of placebo and non-specific therapeutic effects and their ability to change behaviour and produce clinical recovery, and that they can be utilised in therapy to maximise treatment outcomes (Frank, 1982a, 1982b; Walach & Sadaghiani, 2002).

# CHAPTER 5

## 'Conclusions and future directions'

### 5.1 Introduction

Stress is pervasive and can not be avoided in day to day life (Selye, 1976). Under normal circumstances it is not the amount of stress that we face that impairs coping capacity, but rather it is our appraisal of the degree of threat and challenge that is present which leads to stress responses (Lazarus et al., 1985; Morin et al., 2003). In healthy organisms, adaptation occurs when perceived stress is successfully managed, by summoning and using available resources, so that the perceived threat to the self is removed or reduced, and physiological stress responses abate (Lazarus, 1991b, 1993; Lazarus et al., 1985).

In stress related disorders, such as Major Depressive Disorder (MDD), however, which are defined by dysphoria and a negative thinking style (Bisno, 1985; McDermut, Haaga, & Bilek 1997), there is a greater tendency to appraise daily stressors and hassles as threatening (Lazarus, 1991b), which results in more frequent stimulation of arousal systems and leads to an escalating cycle of dysregulation (Chrousos & Gold, 1992; Gold et al., 1988; Post, 1992). As a consequence, circulating stress hormones become chronically elevated. This disrupts normal homeostatic regulation, and has deleterious effects on mood, cognition, behaviour, circadian and diurnal regulation of sleep-wake cycles, and appetitive functions, which can culminate in severe impairment of daily functioning.

As discussed in Chapter 3, depression is the biggest contributor to loss of productivity and personal suffering in the developing world, and is predicted to

increase to epidemic proportions by 2020, with huge costs to society (Brunello et al., 2000; Murray & Lopez, 1997; Murray & Lopez, 1996b). Depression exacts further costs to society as it increases the risk of early death and is a major risk factor in the development of cardiovascular disease (Wulsin, Vaillant, & Wells, 1999). Sleep disturbance is commonly co-morbid with depressive disorders, reflecting the degree of dysregulated arousal, and further contributes to personal suffering (McCrae et al., 2003; Okuji et al., 2002), and increases the risk of mortality for older individuals (Dew et al., 2003). There is an urgent need, therefore, for health initiatives to curtail this global rise in depression in order to lessen the burden of disease.

Contemporary models of depression consistently highlight the involvement of arousal systems and their role in maintaining depressive symptomatology (Chrousos & Gold, 1992; Everly & Benson, 1989). In support of the arousal hypothesis, successful antidepressant treatment has been shown to exert its effects by normalising arousal systems and restoring sleep quality (Barden et al., 1995; Kasckow et al., 2001; Riemann et al., 2001). Newer antidepressant treatments directly target arousal systems and act to dampen stress responses in order to achieve their antidepressant effects (Wolkowitz & Reus, 1999; Zobel et al., 2000). Some people, however, find antidepressant treatment intolerable because of unwanted side effects, or are unable to take antidepressants because of pregnancy or medical illness, and turn to alternative treatments. Relaxation therapy is a widely accepted treatment for stress-related disorders and has been shown to improve long term treatment outcomes when combined with behaviour therapy and problem focussed psychotherapy in the treatment of depression (Krampen, 1999).

Relaxation responses are antithetical to stress reactions as they produce physiological responses that are in direct opposition to stress responses. As outlined in Chapter 3, stress appraisals activate both the hypothalamic-pituitary-adrenal axis (HPA) and the release of norepinephrine from the locus ceoruleus (LC/NE) in the brainstem, in order to mobilise adaptive behaviours necessary to cope with the perceived stress. Specifically, activation of the HPA system leads to the release of corticotrophin releasing hormone (CRH) from the paraventricular nucleus (PVN) in the thalamus, which in turn stimulates the release of adrenocorticotrophin hormone (ACTH) from the adenohypophysis. Adrenocorticotrophin hormone targets the adrenal cortex and stimulates the release of stress hormones, such as glucocorticoids or cortisol, into general circulation. Simultaneously, an immediate alarm reaction is triggered as norepinephrine is released into the bloodstream. As a result, heart rate increases, respiratory rate quickens and breathing becomes shallow, oxygen consumption and metabolic rate increase, and blood flow is redirected to skeletal muscles in readiness for 'fight or flight' (Selye, 1976). Accompanying these physiological changes are feelings of anxiety, apprehension and worry, and increased mental vigilance. Frequent elicitation of relaxation responses has been shown to readjust over-reactive stress responses by favouring activation of inhibitory parasympathetic inputs and dampening HPA activity (Everly & Benson, 1989; Lehrer et al., 2000; Vaschillo et al., 2002).

Relaxation responses can be induced by various methods, such as progressive muscle relaxation (Matsumoto & Smith, 2001; Scheufele, 2000), mental quieting (Benson, 1975), photic stimulation (Brauchli, 1993; Morse, 1993), or voluntary control of breathing, which directly impacts on parasympathetic activity (Vaschillo et al.,

2002). Benson (1975) defined the necessary criteria common to all relaxation methods: the creation of a 'quiet space' free from interruption, the taking up a comfortable position which is conducive to the release of muscle tension, the use of a repetitive word, phrase, or stimulus, designed to focus the mind and distract it from internal dialogue, and above all, the adoption of a 'passive attitude' in order to achieve mental quieting. In contrast to stress responses, relaxation responses decrease heart rate, blood pressure, respiratory rate, and oxygen consumption, cause peripheral vasodilation, promote the redirection of blood flow for digestive and sexual functions, and are usually accompanied by increased well-being and feelings of 'warmth', and 'peacefulness' (Ford et al., 1982; Smith et al., 1996). Frequent elicitation of relaxation responses has been shown to create 'training effects' which blunt reactivity to stressful stimuli (Lucini et al., 1997) and permanently alter mood and physiological functioning, to produce a generalised decrease in arousal (Agras et al., 1980; Ford et al., 1982; Krampen, 1999; Matsumoto & Smith, 2001; Rosen et al., 2000).

Photic stimulation has been shown in this research, and in previous studies (Brauchli et al., 1995; Morse, 1993; Ossebaard, 2000), to reliably induce relaxation responses quickly and effortlessly. Photic stimulation satisfies all of Benson's criteria for inducing relaxation responses. For example, repetitive light flicker provides a stimulus to distract and focus the mind. The wearing of goggles, and headphones if using audiovisual stimulation, act to create a personal space, and reduce environmental influences. In addition to traditional relaxation techniques, photic stimulation utilises specific frequencies of stimulation designed to 'lull' the brain into states conducive to deep relaxation. As a consequence, muscle tension decreases as

relaxation responses are quickly and easily induced, while novel and repetitive light stimuli help to maintain the relaxed state. Photic stimulation also creates altered states of consciousness typically associated with states of deep relaxation (Mundy-Castle, 1953; Walach, 1998). Unlike most relaxation therapies, however, photic stimulation is a passive therapy, is easy to use, and requires no prior learning in order to be effective. For these reasons, photic stimulation was predicted to be more effective than traditional relaxation therapies for a group of depressed individuals, in whom difficulties with motivation and compliance are common.

This thesis draws upon a strong foundation of research which shows that photic stimulation produces photic driving responses in the cortex and can be used to alter cortical activity (Silberstein, 1995b; Toman, 1941; Walter & Walter, 1949). It also rests on a large body of EEG research providing evidence that different EEG rhythms reflect different levels of cortical activity, and are often related to specific mood states (Basar et al., 1997; Brown, 1970; Hinrichs & Machleidt, 1992; Steriade et al., 1990; Sterman, 1996). The underlying assumption in this thesis is that different frequencies of photic stimulation could be used to create specific EEG patterns that in turn, produce related mood and physiological changes. It was anticipated that frequent, long term exposure to entraining stimuli would produce EEG training effects which would be evident as increases in EEG power at entrainment frequencies, with enduring and beneficial effects on mood and sleep.

This research explored the use of brainwave entrainment techniques, and their capacity to influence mood and reduce stress. It adds to previous research as it explored the clinical utility of brainwave entrainment, not just its immediate impact

on arousal and mood, which has been extensively explored (Brauchli et al., 1995; Dieter & Weinstein, 1995; Morse, 1993; Mundy-Castle, 1953). Using a clinical paradigm, two studies tested the effects of regular exposure to auditory and photic stimulation, over an extended period of time, on emotional and physiological regulation.

## 5.2 Study 1

Study 1 addressed the preliminary question, 'whether brainwave entrainment is superior to well established relaxation therapy'. It also explored the effects of particular frequencies of audiovisual stimulation (AVS) on mood and arousal in a non-clinical sample. Consistent with previous research, AVS quickly produced decreases in physiological arousal (Morse, 1994b; Morse & Chow, 1993; Ossebaard, 2000), and was found to be equally as effective as autogenic relaxation at inducing relaxation responses. In this study, however, even the placebo group demonstrated relaxation responses. Therefore, sitting quietly for 20 minutes, with eyes closed, and wearing eye glasses and headphones, but with no stimulation, induced relaxation responses, possibly because they provided most of the necessary requirements for relaxation outlined by Benson (1975). Photic and auditory driving responses were found, and evidenced by moderate effect sizes, however, their detection was hampered because of low statistical power.

These moderate entrainment effects showed that audiovisual stimulation at 5Hz tended to increase EEG magnitude within the 12-14 Hz bandwidth, while stimulation at 22Hz tended to increase energy within the 4-6Hz bandwidth, most likely due to harmonic and sub-harmonic responses. Published research using this data and

applying a non-linear dynamic model, also found entrainment like responses with 22Hz stimulation, but not at the other frequencies (5 & 13Hz) (Gregson & Leahan, 2003). Gregson and Leahan (2003) found that 22Hz AVS intermittently captured the EEG, possibly because of the presence of sub harmonic influences operating closer to the dominant frequency. In addition, Study 1 found that autogenic relaxation tended to decrease theta activity across sessions, possibly due to the increased cortical activity necessary to process the taped instructions.

Mood effects were also present, with trends in predicted directions. Audiovisual stimulation at 13Hz tended to increase subjective relaxation, and enhance positive affect, after a number of sessions, while stimulation at 22Hz tended to increase physiological arousal, and decrease positive affect. As anticipated, negative affect was positively associated with indices of arousal and beta activity, thus supporting earlier work (Bonnet & Arand, 2001; Lim et al., 1996; Nofzinger et al., 2000). Of interest in this non-clinical sample, however, were the findings of significant reductions in depression and anxiety symptoms over the course of the study, thus indicating that brainwave entrainment had the potential to be an effective treatment for depression and arousal disorders. In Study 2 it was anticipated that using a clinically depressed sample, with predicted hyper-arousal and accompanying dysphoria, would reveal stronger treatment responses, as the group had more to gain, and much greater room for improvement.

### 5.3 Study 2

Study 2, extended the findings of study 1, by using a clinically depressed sample, who were hypothesised to be hyper-aroused with disturbed autonomic regulation,

which was reflected in the presence of comorbid sleep disturbance. It also improved on previous research by including a control group, plus a sham condition, the use of a double blind procedure, and random allocation of participants to experimental conditions. Study 2 differed from the first study, in that it attempted to mimic the therapeutic setting, by providing participants with a Lightmask to use at home, for 20 minutes a day, for a period of one month. It was anticipated that this would maximise exposure to entrainment frequencies and relaxation responses, and therefore, facilitate training effects that would be evident as enduring changes in mood and EEG.

Study 2 supported Davidson's model of frontal cerebral asymmetry and emotion, with the finding of increased left midfrontal alpha, relative to the right, in a depressed sample. Davidson postulated that frontal cerebral asymmetry is a diathesis for negative affect and cognitive style, and a trait marker for depression (Davidson, 1998a). Specifically, he proposed that the left frontal cortex is involved in pleasure-seeking or approach type behaviours, while the right frontal cortex is involved in withdrawal behaviours and associated with emotions of fear, anxiety, and disgust (Davidson & Henriques, 2000; Tomarken et al., 1992). Increased left frontal alpha activity indicates deactivation in this area, and therefore, a diminished ability to experience positive emotions, and a predisposition towards negativity (Davidson & Henriques, 2000; Henriques & Davidson, 1990, 1991).

Study 2 also showed that photic stimulation contributed to improvements in sleep, and possibly mood, to a small degree, but unfortunately these effects were indistinguishable from a background of significant treatment effects elicited by non-

specific factors such as demand characteristics, therapeutic effects, and placebo responses. As discussed in Chapter 4, depression research is plagued by the presence of significant placebo responses, especially when mild to moderately depressed samples are used, as was the case in this study (Brown, 1994; Keck, Welge, McElroy, Arnold, & Strakowski, 2000; Kirsch, 2000). Research assessing the effectiveness of psychotherapy for depression shows that a large proportion of treatment responses are obtained in the first few weeks of therapy, before the crucial elements of the therapy have been presented (Ilardi & Craighead, 1994, 1999). Clearly, placebo and non-specific factors contribute to treatment outcomes, with 'early responders' able to maintain treatment gains over time (Haas, Hill, Lambert, & Morrell, 2002). The current study is consistent with these findings, with a substantial drop in depression levels after only 2 weeks of photic stimulation and sleep diary maintenance, which was sustained until follow-up some 10 weeks later.

In summary, both studies showed that brainwave entrainment reliably produced immediate relaxation responses contingent on stimulus presentation. The finding of additional training responses, with enduring effects on EEG and mood, which would be necessary to validate the use of brainwave entrainment in clinical therapy, were not found. These studies are consistent with previous research which found brainwave entrainment to be an effective short term anxiolytic, but unable to produce long term changes in physiological arousal, and emotion regulation (Ossebaard, 2000; Walach, 1998).

Both studies elicited strong placebo responses. As outlined in Chapter 4, Frank (1973, 1982a, b) proposes that clinical therapy fosters placebo responses by

providing a caring therapist, a 'healing setting', a convincing treatment rationale, and a 'ritual' or set of predictable procedures. Both studies provided these features, which, plausibly, contributed to treatment outcomes. In addition, photic stimulation and EEG recording, both of which use sophisticated equipment, may also elicit potent demand characteristics and contribute to placebo responses (Ho et al., 1988). Other research shows that therapeutic touch effectively produces relaxation responses (Gordon, Merenstein, D'Amico, & Hudgens, 1998; Meehan, 1998; Wilkinson et al., 2002). Other studies, however, found no additional effects contributable to therapeutic touch (Engle & Graney, 2000; Shiflett, Nayak, Bid, Miles, & Agostinelli, 2002). Given that both studies included EEG measures, which involved considerable touching of the scalp, this may have contributed to placebo responses. Furthermore, in study 2, which demonstrated stronger placebo responses than study 1, participants were required to keep sleep diaries for up to 6 weeks. For some participants, diary keeping made depressive and sleep symptoms more salient and led to changes in behaviour, which impacted on treatment recovery. According to Ilardi and Craighead (1994) homework can help to increase self-efficacy, which in turn contributes to clinical recovery.

### 5.4 Using placebo to treat depression

An obvious conclusion from this research is that placebo responses are potent treatment tools. Most clinical research such as this attempts to disentangle placebo and treatment effects, by anticipating that treatment responses will be detectable over and above placebo effects. Small to moderate treatment effects are difficult to detect when placebo effects are large, which often occurs in studies assessing therapies for depression, and particularly if studies lack statistical power, as was the case in this

research (Schatzberg & Kraemer, 2000). It has been suggested, however, that placebos are active agents that can be used in the therapeutic setting to maximise treatment outcomes and should not be discarded as peripheral an unimportant (Bench, 2001; Brown, 1994; Frank, 1982a; Goldberg, Weller, & Blittner, 1982; Walach & Sadaghiani, 2002). In study 1, however, the entrainment responses that were present, emerged alongside placebo responses, despite a methodology which acted to conceal them.

## 5.5 Limitations of the research

Study 1 may have been limited in its ability to detect entrainment responses, because of the montage used. Sequential or bipolar montages are superior for filtering out interference, such as environmental noise, which impacts simultaneously across active electrodes and is rejected from the EEG recording because of common mode rejection (Kaiser, 2000; Niedermeyer, 1993; Rosenfeld, 2000). If auditory and photic driving occurred in Study 1, with recruitment of responses across the cortex, it would increase synchrony across sites, thus affecting the cortex in a more global manner. As a consequence, there would be an increase in common activity between active electrode sites, and because of common mode rejection, much of the response would be cancelled out. Despite this, there was evidence of entrainment occurring in Study 1, with moderate non-significant interaction effects showing enhanced activity in the 22Hz and 13Hz bands in response to 22Hz AVS. The fact that entrainment effects were present at all is testimony that AVS did indeed produce entrainment effects in Study 1, which were possibly higher than the data suggest in order for them to be detected at all.

Study 2 addressed these issues and maximised the potential to detect entrainment responses by using a referential montage with a linked ears reference, which more accurately reflects activity at individual electrode sites (Davidson, 1998b; Kaiser, 2000). As a result, very clear photic driving responses were noted, often with harmonic effects, in response to 10Hz stimulation. This study did not support Pockberger's (1985) assertion that photic driving responses are positively related to depression severity, and attenuate on recovery. Rather, photic driving response was unrelated to depression level and negative affect, but appeared to be related to some aspects of physiological arousal. For example, enhanced photic driving response was associated with difficulties with sleep initiation, and decreased sleep efficiency, both of which are accompanied by hyper-arousal.

A limitation of Study 2 was the inability to directly assess physiological arousal. It is possible that the sample chosen was not as hyper-aroused as predicted, despite the presence of sleep disturbance, which is often comorbid with depression because of increased nocturnal cortisol secretion (Nofzinger et al., 2000). This was supported by the finding of low amplitude beta in many participants, but not supported by the high level of comorbid anxiety, which suggests that substantial levels of arousal were indeed present.

Further to this point, in Study 2, photic stimulation enhanced waking mood to a small degree over and above continuous light, thus indicating that photic stimulation affected some aspect of sleep that was not directly assessed in the study. It is possible that photic stimulation induced superior relaxation effects and decreased nocturnal arousal, which impacted on waking mood. In addition, there was a trend

for 5Hz stimulation to decrease sleep onset to a greater degree than other frequencies, which adds further support to this arousal reduction hypothesis. Assessment of salivary cortisol would have shed more light on this issue by providing an objective measure of arousal system functioning. Future research may also consider using polysomnography to see if photic stimulation exerts its effects on waking mood, possibly by reducing nocturnal arousal, and thereby normalising sleep architecture.

Another explanation for the small effects observed on sleep parameters in study 2, particularly sleep onset, is that late evening light tends to decrease melatonin, at a time when it is required for sleep initiation. Most participants reported using their Lightmask before retiring. Reductions in melatonin have been demonstrated following stimulation with low luminance opaque white light (Shealy et al., 1990), however, given that low luminance red light was used in study 2, it is likely that this effect was quite small. Nevertheless, pulsed evening white light tends to phase delay human circadian functioning, which may have a beneficial effect in depression. Depressed individuals often experience shortened latency to REM sleep and decreased slow wave sleep, due to elevated and early secretion of nocturnal cortisol, which greatly undermines sleep quality (Nofzinger et al., 2000). Delaying nocturnal cortisol secretion would assist by increasing the latency to REM sleep, and delaying wake time, therefore improving overall sleep efficiency. Recent research has shown that low luminance (8 Lux) short wavelength light (blue or violet) can influence circadian rhythm function, and offset nocturnal cortisol secretion (Warman et al., 2003). An alternative model for the treatment of depression, therefore, may be to use short wavelength, low luminance light, presented as a light flicker, to combine the

effects of brainwave entrainment, early in the evening, in an attempt to phase delay night time cortisol release, with the effect of increasing the period of slow wave sleep, and thereby improve sleep quality. Regular practice, for a period of longer than 4-5 weeks, would be advised, in order to maximise training effects, as recent research has shown that relaxation responses are greatly augmented if practised for longer than 4-5 weeks (Matsumoto & Smith, 2001). This model may prove to be more beneficial for depressed, hyper-aroused, sleep disturbed individuals.

### 5.6 Future directions

Traditional methods of EEG recording use filters in an attempt to remove unwanted noise and extraneous input from muscle and physiological activity, but in the process can remove potentially relevant information about brain function (Gregson, 2003; Watters, 1999). In addition, Fast Fourier Transformation techniques are applied across numerous data points, in order to derive a mean for a particular bandwidth. Linear methods of statistical analysis, using grouped data, further reduce data variance and can preclude the presence of internal complexities (Gregson, 2003, in press). Non-linear dynamical methods of analysis have the ability to assess the richness and complexity of EEG data which can be lost with traditional linear techniques (Gregson, 2002; Watters, 1999). As discussed in Chapter 4, healthy biological systems are characterised by a wide range of variance and complexity, reflecting their adaptability (Pool, 1989; Winfree, 1987). Reductions in complexity, such as occurs in MDD (Nandrino et al., 1994), with increased frontal lobe alpha, or excessive chaotic behaviour, as is the case in epilepsy (Wright & Liley, 1996), signal diminished adaptive ability and are indicators of disease.

In the current research, photic stimulation influenced the EEG in a complex manner, with harmonic and sub-harmonic responses found. The linear statistical methods used, however, possibly precluded the ability to show the full extent of this complexity over time, which may be remedied using additional non-linear analysis. This research also highlights the problems inherent in assuming that cortical stimulation using a particular frequency will only affect bandwidths at those frequencies. As was shown in this research, and other studies, harmonic and sub-harmonic effects produce complex patterns of EEG response, which in turn can have a range of influences on mood and behaviour (Rosenfeld et al., 1997; Walter & Walter, 1949).

## 5.7 Conclusion

Ample evidence was provided in this thesis to show that depression can be defined as a stress-related disorder, characterised by over stimulation of arousal systems, with accompanying dysphoria and sleep disturbance (Arborelius & Owens, 1999; Nofzinger et al., 2000). Depression is a serious health issue in developing countries, contributing substantially to loss of productivity, and personal suffering, with forecasts of increasing burden and social cost in the future (Murray & Lopez, 1997). There is urgent need, therefore, for effective and easy to use health interventions to help curb this trend.

In response to this need, this project investigated the use of a non-pharmacological treatment, photic stimulation, as an adjunctive therapy in the treatment of MDD. This research replicated earlier work and showed that photic stimulation produces immediate relaxation effects, effortlessly, and without prior training (Brauchli et al.,

1995; Morse, 1993, 1994b; Ossebaard, 2000), but anticipated long term training effects on mood and EEG, as a result of regular, long term use, were not found. This research demonstrated clear frequency following responses in the cortex, with some enhancement to mood and small improvements to sleep quality.

The short term anxiolytic qualities of photic stimulation were demonstrated and remain effective therapeutic tools, possibly more suited for acute pain, or acute stress responses which demand immediate intervention. Arousal models of depression argue that repeated stress responses in depression, act to sensitise arousal systems and increase stress responsivity. As a consequence, each depressive episode leads to an escalating cycle of increasing vulnerability with subsequent depressive episodes becoming more severe and protracted, and less amenable to treatment. To prevent this cycle, it is proposed that stress responses need to be ameliorated quickly and effectively (Everly & Benson, 1989; Post, 1992). Photic stimulation, therefore, is a potent treatment tool for immediate stress management, particularly at a time when coping resources, and motivation, are at their lowest. As a consequence, photic stimulation may work to limit the severity of depressive episodes and help to inhibit the vicious cycle of sensitisation of arousal systems. Photic stimulation may yet help to play a role in ameliorating the burden of disease in depression, and its effects may only be evident over many years, as the severity of depressive episodes is diminished.

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# APPENDIX A

## Study 1 'Thoughts Emotions & Health Questionnaire'.

# Thoughts, Emotions, & Health Questionnaire (p.1).

***Name*** .....***Subject ID***.....

***Address***: .....

***Occupation***.....***Date:*** .....

***Contact numbers: home*** ..... ***work***.....

***Sex***: Male Female ***Age*** .....

***Marital Status*** Single Married

Divorced/ Separated De facto

***Number of children*** .....

**Thank you for volunteering to participate in this research. The following questions ask about your general health, health behaviours, thoughts and feelings. Please read the questions carefully and answer to the best of your ability.**

## FEELINGS & EMOTIONS

This scale consists of a number of words that describe different feelings and emotions. Read each item and then circle the appropriate answer next to the word. Indicate **to what extent you have felt this way in the last WEEK**, that is, how you feel on the average. Use the following scale to record your answers.

| 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|
| **very slightly or not at all** | **a little** | **moderately** | **quite a bit** | **extremely** |

| | | | |
|---|---|---|---|
| **1 2 3 4 5** | interested | **1 2 3 4 5** | irritable |
| **1 2 3 4 5** | distressed | **1 2 3 4 5** | alert |
| **1 2 3 4 5** | excited | **1 2 3 4 5** | ashamed |
| **1 2 3 4 5** | upset | **1 2 3 4 5** | inspired |
| **1 2 3 4 5** | strong | **1 2 3 4 5** | nervous |
| **1 2 3 4 5** | guilty | **1 2 3 4 5** | determined |
| **1 2 3 4 5** | scared | **1 2 3 4 5** | attentive |
| **1 2 3 4 5** | hostile | **1 2 3 4 5** | jittery |
| **1 2 3 4 5** | enthusiastic | **1 2 3 4 5** | active |
| **1 2 3 4 5** | proud | **1 2 3 4 5** | afraid |

## THOUGHTS (p.2)

Listed below are a variety of thoughts that pop into people's heads. Please read each thought and **indicate how frequently, if at all, the thought occurred to you over the last WEEK** by circling one response. Please read each item carefully and fill in the appropriate answer by using the following scale:

| 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|
| **not at all** | **sometimes** | **moderately** | **often** | **all the time** |

| Response | Thoughts |
|---|---|
| 1 2 3 4 5 | I feel like I'm up against the world. |
| 1 2 3 4 5 | I'm no good. |
| 1 2 3 4 5 | I am respected by my peers. |
| 1 2 3 4 5 | I have a good sense of humour. |
| 1 2 3 4 5 | Why can't I ever succeed. |
| 1 2 3 4 5 | No one understands me. |
| 1 2 3 4 5 | My future is bright. |
| 1 2 3 4 5 | I will be successful. |
| 1 2 3 4 5 | I've let people down. |
| 1 2 3 4 5 | I am fun to be with. |
| 1 2 3 4 5 | I am in a great mood. |
| 1 2 3 4 5 | I don't think I can go on. |
| 1 2 3 4 5 | I wish I were a better person. |
| 1 2 3 4 5 | There are many people who care about me. |
| 1 2 3 4 5 | I'm proud of my accomplishments. |
| 1 2 3 4 5 | I'm so weak. |
| 1 2 3 4 5 | My life's not going the way I want it to. |
| 1 2 3 4 5 | I will finish what I start. |
| 1 2 3 4 5 | I have many good qualities. |
| 1 2 3 4 5 | I'm so disappointed in myself. |
| 1 2 3 4 5 | Nothing feels good anymore. |
| 1 2 3 4 5 | I am comfortable with life. |
| 1 2 3 4 5 | I have a good way with others. |
| 1 2 3 4 5 | I can't stand this anymore. |
| 1 2 3 4 5 | I can't get started. |
| 1 2 3 4 5 | I am a lucky person. |
| 1 2 3 4 5 | I have friends who support me. |
| 1 2 3 4 5 | What's wrong with me? |
| 1 2 3 4 5 | I wish I were somewhere else. |
| 1 2 3 4 5 | Life is exciting. |

| Response | Thoughts |
|---|---|
| 1 2 3 4 5 | I enjoy a challenge. |
| 1 2 3 4 5 | I can't get things together. |
| 1 2 3 4 5 | I hate myself. |
| 1 2 3 4 5 | My social life is terrific. |
| 1 2 3 4 5 | There's nothing to worry about. |
| 1 2 3 4 5 | I'm worthless. |
| 1 2 3 4 5 | Wish I could just disappear. |
| 1 2 3 4 5 | I'm so relaxed. |
| 1 2 3 4 5 | My life is running smoothly. |
| 1 2 3 4 5 | What's the matter with me? |
| 1 2 3 4 5 | I'm a loser. |
| 1 2 3 4 5 | I'm happy with the way I look. |
| 1 2 3 4 5 | I take good care of myself. |
| 1 2 3 4 5 | My life is a mess. |
| 1 2 3 4 5 | I'm a failure. |
| 1 2 3 4 5 | I deserve the best in life. |
| 1 2 3 4 5 | Bad days are rare. |
| 1 2 3 4 5 | I'll never make it. |
| 1 2 3 4 5 | I feel so helpless. |
| 1 2 3 4 5 | I have many useful qualities. |
| 1 2 3 4 5 | There is no problem that is hopeless. |
| 1 2 3 4 5 | Something has to change. |
| 1 2 3 4 5 | There must be something wrong with me. |
| 1 2 3 4 5 | I won't give up. |
| 1 2 3 4 5 | I state my opinions with confidence. |
| 1 2 3 4 5 | My future is bleak. |
| 1 2 3 4 5 | It's just not worth it. |
| 1 2 3 4 5 | I can't finish anything. |
| 1 2 3 4 5 | My life keeps getting better. |
| 1 2 3 4 5 | Today I've accomplished a lot. |

## HASSLES (p.3)

This next part of the questionnaire lists areas in your life which may or may not be a problem or a hassle for you. A **HASSLE** is an irritant which annoys or bothers you; they can make you upset or angry, they can be minor irritants or fairly major pressures. Please indicate if any of these following **AREAS** is **currently** a **HASSLE** for you by circling the appropriate response.

**0 None or not Applicable** **1 Somewhat** **2 Quite a bit** **3 A great deal**

| | | | |
|---|---|---|---|
| 0 1 2 3 | Illness of a family member. | 0 1 2 3 | Concerns about weight. |
| 0 1 2 3 | Your partner.. | 0 1 2 3 | Preparing meals. |
| 0 1 2 3 | The weather | 0 1 2 3 | Too many things to do. |
| 0 1 2 3 | Fertility issues. | 0 1 2 3 | Job dissatisfaction. |
| 0 1 2 3 | Your friends. | 0 1 2 3 | Legal matters. |
| 0 1 2 3 | Your children. | 0 1 2 3 | Problems with divorce or separation. |
| 0 1 2 3 | Your parents. | | |
| 0 1 2 3 | Work or study colleagues. | 0 1 2 3 | Concerns about inner |
| 0 1 2 3 | Concerns about money. | 0 1 2 3 | Conflicts. |
| 0 1 2 3 | Use of drugs or alcohol. | 0 1 2 3 | Sex. |
| 0 1 2 3 | Care for pet. | 0 1 2 3 | Concerns about getting ahead. |
| 0 1 2 3 | Trouble making decisions. | | |
| 0 1 2 3 | Home maintenance. | 0 1 2 3 | Employment. |
| 0 1 2 3 | Being lonely. | 0 1 2 3 | Not enough time. |
| 0 1 2 3 | Concerns about medical treatment. | 0 1 2 3 | Transportation problems. |
| | | 0 1 2 3 | Crime. |
| 0 1 2 3 | Physical appearance. | 0 1 2 3 | Political or social issues. |
| 0 1 2 3 | Concerns about health in general. | 0 1 2 3 | Other, please specify ..... |
| 0 1 2 3 | Your environment (noise, space, greenery etc.) | | |

## HEALTH MATTERS.

*Please respond to the following questions by placing a tick in the appropriate box*

In the **past year** have you suffered from a medical condition which has resulted in you being hospitalised? Yes No

If yes, what was the nature of this condition?.....

In the last **3 months** has there being any **event** which has had **a major impact** on your life?

Yes No

If yes, would you please describe the event?.....

## HEALTH BEHAVIOURS (p.4)

### ALCOHOL AND DRUGS

**How often** do you normally drink ALCOHOL? everyday or most days
2-3 times per week
less than 2 times per week
never

**How much** do you normally drink in any one session? 1-2 drinks
3-6 drinks
more than 6 drinks

Do you smoke **TOBACCO**? Yes No

**If yes,**
On average, **how many** cigarettes per day do you smoke? .....

Are you currently taking any **PRESCRIBED MEDICATION**? Yes No
**If yes**, please indicate below the type of medication you are taking and the dosage:

| **Drug** | **Dosage** |
|---|---|
| ---------- | ---------- |
| ---------- | ---------- |
| ---------- | ---------- |
| ---------- | ---------- |

Do you use **RECREATIONAL DRUGS** Yes No
(for example; marijuana, speed, crack, heroin, etc.)

**If yes,**
What do you use?.....

How often would you use this/these drug(s)? everyday or most days
1-2 times per week
less than once per week
less than once per month

## HOW DO YOU FEEL RIGHT NOW? (p.5)

On a scale of **1**(feeling very relaxed and calm) to **10** (feeling very anxious and uptight), please indicate, by circling one number only, **how relaxed and calm or anxious and uptight you feel right now**.

| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|
| very relaxed & calm | | | | | | | | very uptight & anxious | |

## For Women Only:

Because feelings and emotions are affected by different stages of the menstrual cycle would you please indicate what stage of your cycle you are at:

First week (menstruation) .....*Day1-7*

Second week .....*Day 8-14*

Third week .....*Day 15-21*

Fourth week .....*Day22 to end of cycle*

No longer have periods

What is the average length (in days) of your menstrual cycle? .....

**If you have any questions, please don't hesitate to ask.**

**THANK YOU FOR YOUR COOPERATION AND PARTICIPATION.**

NOTES:.....

.....

.....

.....

.....

.....

.....

.....

*Research by Kerry Leahan, Australian National University, approval No: P9706*

# APPENDIX B

Study 1 autogenic relaxation script.

## STUDY 1 RELAXATION SCRIPT.

Ensure that you are comfortable. Scan your body from your head to your toes noticing areas of calm and relaxation (4)….and areas of tension and pressure (8). Adjust your body letting go of tension and ensuring that you are comfortable and relaxed.

Take your focus to your breath. …Notice the air as it passes in and out of your nostrils. Feel the coolness of the air as you breath in ….and the warmth of the air as you breath out. ….*(16)* As you breath in, notice where your breath goes in your chest; is it shallow? …Or is it deeper into the abdomen? …Spend some moments observing your breath. *(16).*

If your mind wanders, and you are distracted by thoughts, quietly guide your awareness back to your breath and let whatever thoughts arise waft past you, as if you can observe them from afar.

Gently and effortlessly, start to slow down your breathing. Take your breath lower into your abdomen. As you breath in let your abdomen fill and expand …(4) and as you breath out, let the abdomen fall. Allow the breath to be easy and effortless. Breath in to the count of 4, …hold the breath for 2 counts, …breath out to the count of 4. Do this for several breaths *(16).* Gently let go of counting and allow the breath to be easy and effortless.

Take your awareness to your head and neck. Notice any areas of tension, and areas of quiet and calm. Now focus on the muscles around your eyes. Notice where there is tension, where there is calm. Let go of any tension and allow the eyes to become soft in their sockets (8). Next become aware of your forehead and your scalp, … notice where there is tightness and where there is quiet and calm (4). Let go of any tightness and allow the muscles to soften and relax (8). Now become aware of your face, jaw, and throat…Allow the muscles to soften, letting go of tightness and tension (4). Feel the whole head as heavy, warm and relaxed. Say to yourself, several times, "my head, neck and throat are calm and relaxed" Rx3 (16).

Allow the heaviness of relaxation to spread to your shoulders. Become aware of the muscles in your shoulders letting go of any tightness and allowing your shoulders to drop and relax. …Say to yourself 'my shoulders are heavy and relaxed' Rx3 (4).

Imagine this feeling of calm and relaxation spreading down your arms. Imagine any areas of tension softening and relaxing as your arms become warm and heavy. Allow the feeling of relaxation to move down your arms, from your shoulders to your elbows, down into your wrists, your hands and to your fingers. Relax your hands and fingers feeling the warmth and heaviness spreading to the tips of your fingers. Say slowly to yourself; several times "my hands are warmer and warmer" (16).

Feel your whole head,… your shoulders, …arms … hands …and fingers as relaxed, warm and heavy. *(8)*

Take your awareness to your chest and abdomen. Notice first the muscles in your chest, if there is any tension and tightness, where there are quiet calm areas (8). Then focus on the muscles of your stomach and abdomen. Notice tension and tightness, notice quiet calm areas. Gently let the edges of any tension or tightness soften now, soften gradually more and more, thinking to yourself: "the muscles of my chest and stomach are quiet and calm" R x3 (16).

Become aware of your pelvis and hips. Notice any areas of tension or discomfort. Imagine that you can breathe into these areas of tightness and discomfort …letting go with each out breath. With each breath you become more relaxed,…more calm,… your whole abdomen and pelvis feels warm, comfortable, and relaxed (4). Say to yourself over and over, "the muscles of my chest, abdomen and pelvis are calmer and calmer" (16).

Allow this feeling of warmth and calm to flow down to your thighs. Allow the muscles of your thighs to become loose, and relaxed. …Let go of any areas of tension. Allow the feeling of warmth and relaxation to filter down from your thighs, into your knees, and on down into your calves and shins, down to your ankles, down to your feet and into the tips of your toes. Both your legs feel heavy, warm, and comfortable. Think to yourself; "the muscles of my legs and feet are relaxed and calm" (16).

Scan your whole body from your toes to your head (8). Gently let go of any remaining tension. Let the outbreath help you to relax, and allow the feeling of relaxed warmth to fill your entire body (8).

…say to yourself 'my whole body is relaxed and warm' Rx3.

Take your awareness back to your breath. Notice the breath in your chest. Is it shallow or deep? (4). Observe the rate of your breathing, it is slow and rhythmic or fast and shallow? (8). As you breathe in, observe the coolness of the air in your nostrils …and the warmth of the air as you breath out. (16).

Gently now begin to bring you awareness back to the room and be aware of your body in contact with the chair. (4). Be aware of the noises you can hear in the room. (4) Take several minutes and gently bring yourself back fully to the room feeling your body supported by the chair you are resting on.

# APPENDIX C

Means and standard deviations for Study 1 variables.

Appendix C: Means and standard deviations (SD) for Study 1 variables; heart rate, skin conductance, subjective relaxation, positive affect, negative affect, and EEG amplitude in the 4-6hz, 12-14Hz, and 21-23Hz bandwidths.

**Mean heart rate and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 64.8 (6.4) | 67.9 (10.5) | 69.1 (14.5) | 67.1 (6.9) | 70.7 (9.3) | 69.4 (14.4) | 65.9 (7.4) | 67.2 (9.1) | 67.8 (7.0) | 68.3 (7.0) | 70.7 (10.8) | 68.6 (11.1) | 68.5 (10.6) | 66.8 (11.2) |
| relaxation | 69.0 (12.6) | 70.9 (11.0) | 72.5 (12.8) | 73.5 (11.2) | 68.7 (10) | 74.8 (15.3) | 70 (10) | 75.9 (14.8) | 73.1 (11.1) | 70 (12.8) | 70.1 (11.3) | 74 (10.8) | 76 (16.8) | 68 (8.6) |
| theta | 64.2 (8.5) | 73.4 (16.4) | 71.8 (10) | 68.2 (13.6) | 69.9 (11.6) | 68.3 (12.9) | 73.6 (15.7) | 71.1 (14.3) | 73.7 (9.4) | 69.5 (12) | 69.9 (8.2) | 68.5 (5.2) | 72 (6.5) | 72 (9.9) |
| alpha | 60.9 (11.2) | 65.8 (6.9) | 68.4 (10.6) | 62.2 (6.1) | 70 (8.8) | 70.3 (10.9) | 69.9 (6.8) | 67 (6.5) | 68.2 (7.3) | 67.9 (6.2) | 62.6 (6.8) | 67.8 (4.9) | 65.8 (4.1) | 63.7 (5.9) |
| beta | 62.2 (9.2) | 72.5 (12.1) | 67 (13.7) | 65.9 (9.0) | 65 (9.0) | 71.2 (9.7) | 66.1 (5.1) | 64 (9.0) | 63.4 (5.3) | 64.3 (9.7) | 66.9 (9.6) | 61.7 (7.6) | 63.3 (9.5) | 63.9 (6.6) |

**Mean skin conductance (μmhos) and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 8.9 (4.8) | 10.7 (8.8) | 10.8 (3.5) | 8.7 (5.1) | 7.7 (3.3) | 9.4 96.9) | 8.6 (3.9) | 7.5 (4.4) | 7.8 (4.4) | 10.3 (3.3) | 11.3 (12) | 9.2 (1.3) | 6.9 (3.6) | 10.4 (5.5) |
| relaxation | 9.6 (3.1) | 8.0 (5.1) | 6.3 (4.4) | 9.8 (5.4) | 9.2 (6.0) | 9.3 (7.0) | 10.2 (3.8) | 10.7 (4.8) | 7.8 (2.5) | 12.1 (3.1) | 6.4 (2.9) | 7.9 (2.7) | 7.3 (3.2) | 10.4 (7.6) |
| theta | 10 (6.10 | 13.2 (9.3) | 3.3 (1.1) | 8.6 (5.6) | 12 (9.70 | 12.1 (12.4) | 13.6 (13.1) | 9.0 (5.5) | 8.2 (4.8) | 8.0 (7.5) | 8.5 (4.0) | 12.1 (11.8) | 8.4 (7.4) | 6.5 (2.6) |
| alpha | 6.8 (4.4) | 9.4 (6.8) | 6.5 (2.1) | 5.2 (1.3) | 1.08 (6.5) | 13.4 (10.5) | 7.5 (12.1) | 9.9 (7.5) | 10.1 (11.1) | 6.7 (3.1) | 5.4 (2.9) | 5.9 (3.8) | 10.6 (8.7) | 9.0 (6.3) |
| beta | 13.8 (7.10 | 19.3 .(13) | 16.1 (8.5) | 15.1 (13.7 | 13.6 (7.4) | 16.6 (9.1) | 14.1 (7.3) | 14.7 (5.2) | 11.3 (4.8) | 13.3 (6.9) | 16.5 (8.5) | 11.7 (5.5) | 11.0 (4.5) | 19.1 (5.8) |

Appendix C continued.

**Mean subjective relaxation and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 7.3 (1.2) | 7.8 (2.4) | 8.0 (1.4) | 8.3 (1.0) | 8.2 (1.0) | 7.8 (1.3) | 8.3 (1.4) | 7.7 (1.5) | 7.3 (1.8) | 7.8 (2.5) | 8.2 (1.5) | 7.7 (1.0) | 7.5 (2.5) | 7.7 (1.9) |
| relaxation | 5.3 (0.8) | 6.7 (2.2) | 7.5 (1.6) | 7.7 (1.9) | 7.5 (1.5) | 7.5 (1.5) | 8.0 (1.4) | 8.2 (1.2) | 8.0 (1.5) | 7.3 (1.9) | 8.3 (1.2) | 8.0 (1.7) | 7.7 (1.5) | 7.0 (2.2) |
| theta | 7.3 (1.6) | 6.8 (1.7) | 6.7 (1.9) | 7.2 (1.8) | 7.8 (2.0) | 7.5 (2.9) | 7.7 (2.5) | 8.1 (1.0) | 7.7 (1.9) | 7.7 (2.3) | 8.0 (1.5) | 8.2 (1.7) | 8.3 (1.4) | 8.2 (1.0) |
| alpha | 8.3 (0.5) | 9.2 (0.7) | 9.3 (1.6) | 9.3 (0.5) | 9.3 (0.5) | 9.5 (0.8) | 9.5 (0.5) | 9.3 (0.8) | 8.8 (1.0) | 9.7 (0.5) | 9.7 (0.8) | 9.3 (0.5) | 9.3 (1.2) | 9.0 (0.6) |
| beta | 7.0 (2.4) | 8.0 (1.5) | 7.8 (1.9) | 8.5 (0.5) | 7.8 (1.2) | 7.0 (2.0) | 8.2 (1.6) | 8.0 (0.9) | 7.8 (1.5) | 7.5 (1.5) | 8.2 (1.5) | 7.8 (1.2) | 7.8 (1.2) | 7.8 (1.0) |

**Mean positive affect and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 38 (2.9) | 32 (7.4) | 28.3 (5.5) | 31.8 (10.8) | 28.3 (10) | 27.2 (5.4) | 26.3 (3.3) | 26.6 (7.5) | 29 (5.9) | 28.3 (4.6) | 23.7 (7.8) | 28.0 (7.5) | 26.7 (6.2) | 29.5 (7.9) |
| relaxation | 35 (7.6) | 34.6 (9.3) | 29.5 (8.7) | 31.3 (11.3) | 29.2 (10.7) | 26.5 (12.3) | 27 (10.7) | 28.3 (13.1) | 31 (14.2) | 28.3 (11.5) | 30.2 (12.8) | 26.7 (13.0) | 27.0 (10.8) | 30.7 (12.9) |
| theta | 39 (7.0) | 32.3 (9.0) | 31.8 (11.4) | 28.5 (8.7) | 26.2 (9.5) | 24.8 (10.4) | 26 (8.8) | 29.3 (10.4) | 27.8 (8.0) | 29.6 (8.4) | 28.3 (9.6) | 28.7 (10.1) | 28.3 (11.3) | 32.8 (13.0) |
| alpha | 39 (7.0) | 35.6 (9.4) | 32.5 (5.7) | 32.5 (7.9) | 36.3 (8.1) | 37.5 (10.5) | 37.2 (10.6) | 36.3 (11.2) | 35 (10.4) | 35.5 (11.0) | 36.0 (10.1) | 33.8 (12.0) | 30.3 (13.2) | 33.8 (11.2) |
| beta | 28.8 (7.4) | 25.8 (10.6) | 22.8 (8.3) | 20.2 (9.7) | 20 (11) | 22.3 (9.9) | 19.8 (9.4) | 19.6 (10.1) | 21 (11.2) | 20.5 (10.4) | 22.5 (12.9) | 23.7 (13.1) | 21.7 (9.2) | 24.5 (12.1) |

Appendix C continued.

**Mean negative affect and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 16.7 (3.9) | 20.0 (8.6) | 16.5 (7.3) | 17.0 (4.8) | 17.2 (6.2) | 13.2 (3.1) | 13.8 (2.1) | 15.7 (5.1) | 15.8 (4.8) | 12.8 (3.8) | 15.7 (5.6) | 13.7 (3.3) | 13.7 (3.5) | 13.3 (3.6) |
| relaxation | 19.5 (6.6) | 17.3 (5.0) | 15.8 (4.0) | 13.7 (3.2) | 13.3 (3.1) | 11.8 (1.3) | 11.8 (2.6) | 12.0 (2.4) | 12.7 (3.1) | 13.7 (4.5) | 12.5 (2.7) | 11.2 (1.6) | 12.7 (1.9) | 13.8 (2.0) |
| theta | 19.3 (5.8) | 12.5 (2.0) | 13.0 (4.4) | 14.8 (3.2) | 14.0 (4.0) | 17.8 (8.0) | 13.2 (3.5) | 16.0 (5.4) | 15.2 (6.1) | 13.5 (3.6) | 14.7 (3.4) | 13.3 (4.0) | 12.0 (3.6) | 16.2 (9.0) |
| alpha | 13.8 (3.9) | 16.8 (6.3) | 13.0 (3.7) | 15.3 (4.1) | 13.7 (3.1) | 12.3 (3.4) | 12.7 (3.3) | 16.7 (4.4) | 14.5 (4.2) | 13.7 (3.3) | 12.2 (2.9) | 12.3 (3.2) | 14.7 (5.4) | 15.5 (3.6) |
| beta | 20.0 (5.7) | 18.7 (8.1) | 14.3 (5.2) | 15.8 (6.3) | 14.0 (9.4) | 15.0 (8.0) | 15.3 (5.9) | 16.8 (9.3) | 14.7 (5.0) | 16.8 (8.4) | 17.0 (10.2) | 14.7 (3.9) | 13.5 (3.0) | 16.8 (6.6) |

**Mean occipital 4-6Hz (μV) and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 2.7 (1.0) | 2.2 (1.1) | 2.1 (1.1) | 1.6 (0.6) | 2.2 (0.8) | 2.3 (1.0) | 2.7 (1.1) | 2.0 (1.0) | 2.3 (0.8) | 2.3 (0.9) | 2.2 (0.4) | 2.5 (1.2) | 2.0 (0.6) | 2.3 (1.1) |
| relaxation | 1.8 (0.8) | 1.4 (1.0) | 1.1 (1.2) | 1.2 (0.8) | 1.5 (0.8) | 1.1 (0.5) | 0.9 (1.0) | 0.8 (0.4) | 1.2 90.7) | 1.3 (0.7) | 1.2 (0.8) | 1.2 (0.6) | 1.2 (0.5) | 1.9 (0.8) |
| theta | 2.1 (0.8) | 2.1 (0.5) | 2.0 (0.6) | 2.0 (0.6) | 1.6 (0.9) | 1.5 (0.5) | 2.2 (0.8) | 2.4 (0.7) | 2.3 (0.7) | 2.3 (0.8) | 2.3 (0.7) | 2.3 (0.6) | 2.5 (0.9) | 2.9 (1.3) |
| alpha | 2.1 (1.0) | 1.2 (0.6) | 1.7 (0.9) | 1.7 (0.8) | 1.3 (0.6) | 1.3 (0.9) | 1.8 (0.6) | 1.3 (0.5) | 1.7 (1.1) | 1.7 (0.7) | 1.9 (0.8) | 2.1 (0.6) | 2.0 (1.0) | 1.9 (0.5) |
| beta | 1.6 (0.4) | 1.5 (0.7) | 1.5 (0.4) | 2.2 (1.6) | 2.1 (1.2) | 2.4 (2.2) | 2.5 (2.3) | 1.5 (0.4) | 2.0 (1.3) | 1.7 (0.3) | 1.5 (0.4) | 1.5 (0.3) | 1.6 (0.5) | 1.5 (0.3) |

Appendix C continued.

**Mean occipital 12-14Hz (μV) and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 1.6 (0.5) | 1.3 (0.3) | 1.4 (0.5) | 1.0 (0.4) | 1.5 (0.7) | 1.6 (0.7) | 1.6 (0.7) | 1.5 (0.7) | 1.5 (0.3) | 1.4 (0.2) | 1.4 (0.2) | 1.5 (0.6) | 1.4 (0.2) | 1.5 (0.2) |
| relaxation | 1.4 (0.6) | 1.4 (1.0) | 1.1 (1.2) | 1.1 (0.8) | 1.2 (0.6) | 1.0 (0.7) | 0.7 (0.9) | 0.8 (0.5) | 1.1 (0.7) | 1.2 (0.7) | 1.2 (0.8) | 0.9 (0.6) | 1.2 (0.6) | 1.5 (0.8) |
| theta | 1.8 (0.8) | 2.1 (0.9) | 1.9 (1.3) | 1.8 (1.0) | 1.9 (1.4) | 1.5 (0.9) | 1.9 (1.1) | 2.3 (1.2) | 1.9 (0.9) | 2.0 (1.0) | 1.8 (0.7) | 1.7 (0.6) | 2.0 (0.8) | 1.7 (0.4) |
| alpha | 1.2 (0.3) | 1.0 (0.7) | 1.4 (0.9) | 1.4 (0.8) | 1.4 (1.0) | 1.1 (0.6) | 1.6 (0.8) | 1.1 (0.4) | 1.3 (0.6) | 1.3 (0.3) | 1.6 (0.8) | 1.7 (0.6) | 1.8 (0.6) | 1.2 (0.2) |
| beta | 1.2 (0.4) | 1.1 (0.4) | 1.2 (0.5) | 1.5 (0.9) | 1.3 (0.6) | 1.7 (1.3) | 1.8 (1.3) | 1.1 (0.3) | 1.2 (0.5) | 1.3 (0.3) | 1.1 (0.4) | 1.1 (0.3) | 1.2 (0.4) | 1.0 (0.2) |

**Mean occipital 21-23Hz (μV) and (SD) for control, relaxation, theta, alpha and beta groups (n=30)**

| session | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| control | 0.7 (0.2) | 0.6 (0.2) | 0.7 (0.2) | 0.5 (0.2) | 0.7 (0.4) | 0.7 (0.2) | 0.7 (0.1) | 0.6 (0.2) | 0.7 (0.2) | 0.7 (0.2) | 0.6 (0.1) | 0.7 (0.2) | 0.6 (0.1) | 0.7 (0.1) |
| relaxation | 0.8 (0.2) | 0.7 (0.3) | 0.5 (0.3) | 0.4 (0.2) | 0.7 (0.3) | 0.6 (0.3) | 0.4 (0.3) | 0.4 (0.2) | 0.8 (0.6) | 0.8 (0.4) | 0.7 (0.6) | 0.6 (0.3) | 0.7 (0.6) | 0.7 (0.1) |
| theta | 0.8 (0.3) | 0.8 (0.4) | 0.7 (0.3) | 0.6 (0.2) | 0.6 (0.4) | 0.5 (0.2) | 0.8 (0.5) | 0.8 (0.3) | 0.7 (0.3) | 0.7 (0.3) | 0.7 (0.3) | 0.7 (0.2) | 0.9 (0.3) | 0.8 (0.3) |
| alpha | 0.6 (0.2) | 0.4 (0.1) | 0.6 (0.4) | 0.6 (0.4) | 0.5 (0.4) | 0.5 (0.3) | 0.7 (0.3) | 0.5 (0.1) | 0.5 (0.1) | 0.6 (0.2) | 0.7 (0.3) | 0.7 (0.2) | 0.8 (0.4) | 0.7 (0.3) |
| beta | 0.5 (0.1) | 0.5 (0.2) | 0.5 (0.1) | 0.6 (0.3) | 0.6 (0.3) | 0.7 (0.4) | 0.8 (0.5) | 0.5 (0.1) | 0.6 (0.2) | 0.5 (0.1) | 0.5 (0.1) | 0.5 (0.1) | 0.5 (0.2) | 0.5 (0.1) |

# APPENDIX D

Ranges of Pearson's correlation coefficients for Study 1 variables.

Appendix D: Ranges for Pearson's correlation coefficients for study 1 variables across 14 sessions.

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 global theta | **1.0** | | | | | | | | | | | | | |
| 2 global alpha | [.50,.**85****] | **1.0** | | | | | | | | | | | | |
| 3 global beta | [.20,.**80****] | [.**48****,.**81****] | **1.0** | | | | | | | | | | | |
| 4 heart rate | [.07,-.**40***] | [-.02,.**38***] | [01,**50***] | **1.0** | | | | | | | | | | |
| 5 skin conductance | [-.20,.**58****] | [-.03,.**43***] | [-.01,**46***] | [-.04,.**52***] | **1.0** | | | | | | | | | |
| 6 subjective relaxation | [-.01,.**40***] | [-.01,.**43***] | [-.03,.**38***] | [-.35,-.04] | [-.30,-.03] | **1.0** | | | | | | | | |
| 7 depression | [.04,.05] | [.06,.09] | [.12,.14] | [-.03,.10] | [-.01,.01] | [-.06,-.30] | **1.0** | | | | | | | |
| 8 anxiety | [.06,.15] | [-.03,.10] | [-.05,.20] | [-.07,.16] | [.10,.30] | [-.**45***,.**42***] | [.**60***,.**70****] | **1.0** | | | | | | |
| 9 global severity index | [-.07,.08] | [.02,.03] | [-.07,.13] | [.03,.17] | [-.09,.23] | [-.**38***,-.13] | [.**78****,.**80****] | [.**70****,.**80****] | **1.0** | | | | | |
| 10 positive affect | [-.**40***,.01] | [-.**38***,-.01] | [-.02,.32] | [-.25,.24] | [-.21,.25] | [.07,.25] | [-.**43***,-.30] | [-.33,-.20] | [-.**44***,-.**41***] | **1.0** | | | | |
| 11 negative affect | [.01,.13] | [-.30,.30] | [-.33,.**42***] | [-.20,.**37***] | [-.17,.**39***] | [-.**49***,-.07] | [.16,.**65****] | [.**40***,.**72****] | [.02,.**69****] | [.03,-.**57****] | **1.0** | | | |
| 12 positive thoughts | [15,30] | [-.27,10] | [.14,.19] | [-.03,-.01] | [.01,.05] | [.16,.21] | [.05,-.**36***] | [.02,-.25] | [-.**40***,-.10] | [.**70****,.**80****] | [-.33,.12] | **1.0** | | |
| 13 negative thoughts | [.13,.17] | [-.05,.11] | [-.02,.10] | [-.26,.14] | [-.03,.01] | [-.20,.35] | [.**50***,.**70****] | [.25,.**52****] | [.**40***,.**60****] | [-.**50****,-.05] | [-.20,.**60****] | [-.13,.06] | **1.0** | |
| 14 daily hassles | [-.20,-.03] | [-.28,27] | [.07,.35] | [.08,.**42***] | [-.01,.29] | [-.**62***,01] | [**46***,**57***] | [30,**39***] | [.35,.**53***] | [-.12,.28] | [.30,.**56***] | [-.05,.17] | [.22,.**70****] | **1.0** |
| 15 menstrual cycle | [-.**50****,-.02] | [-.**46***,.12] | [-.32,.16] | [.01,.30] | [-.30,.18] | [-.30,.27] | [-.30,-.27] | [-.18,-.17] | [-.32,-.31] | [.07,.**55****] | [-.**41***,.26] | [-.01,.14] | [-.22,-.17] | [-.31,-.07] |

* significance LE .05 ** significance LE .01

# APPENDIX E

Study 2 Selection Criteria.

# SELECTION CRITERIA:

THE USE OF PHOTIC STIMULATION TO TREAT MOOD AND SLEEP PROBLEMS

Participants will be eligible to enter the study if they have **mild or moderate depression** and **sleep disturbance**. Please use the following criteria to guide your selection.

People **will not be admitted** to the study if they:

- Have a history of **bipolar disorder** or are **psychotic**.
- Have a history of **epilepsy** or **migraine.**
- Are currently taking **anti-depressant**, or **anxiolytic** medication.
- Report **snoring** most nights or 'more nights than not'.

***1. Criteria for Depression***

Presence of at least Mild Depression which includes at least **1 of the first two symptoms** and at least **4 of the others** occurring over the last two weeks.

1. Two weeks of abnormal depressed mood (feeling sad most of the time) ❑
2. Loss of interest and/or pleasure..... ❑
3. Change in appetite (weight loss or weight gain)..... ❑
4. Sleep disturbance (insomnia or hypersomnia most days)..... ❑
5. Psychomotor agitation or retardation (observable by others) ..... ❑
6. Fatigue or loss of energy (most days) ..... ❑
7. Feelings of worthlessness or guilt. ..... ❑
8. Poor concentration, difficulty thinking & or memory disturbance ..... ❑
9. Recurrent thoughts of death ..... ❑
10. Above symptoms cause distress & or impairment in functioning ..... ❑

***2. Criteria for sleep disturbance***

History of sleep disturbance for about one month, including some of the following symptoms:

In general describes sleep as poor. ..... ❑
In general, wakes feeling tired and not refreshed after a nights sleep. ..... ❑
Difficulty falling asleep..... ❑
Wakes several times during the night. ..... ❑
Difficulty falling asleep after waking- toss and turn, lie there worrying. .. ❑
Insomnia. ..... ❑
Hypersomnia (excessive sleepiness). ..... ❑
History of night sweats. ..... ❑
History of night mares/night terrors. ..... ❑
Thrashes about when sleeping. ..... ❑
Daytime fatigue and/or sleep episodes..... ❑

# APPENDIX F

Study 2 'Health Matters Questionnaire'.

THE USE OF PHOTIC STIMULATION TO TREAT MOOD AND SLEEP PROBLEMS

HEALTH MATTERS QUESTIONNAIRE (p.1)

***Name*** .....

***Address***: .....

***Occupation*** ..... ***Date:*** .....

***Contact numbers: home*** ..... ***work*** .....

***Other:*** .....

***Sex***: Male ❑ Female ❑

***Date of Birth*** ..... ***Age***

***Health Professional:***
In the event that your depression gets worse over the course of the study could you please give us the name of a health professional we can refer you to:

***General Practitioner*** ..... Phone number .....

Please indicate any other health professionals involved in your care, such as Psychologist, Psychiatrist or Case Manager:

.....

.....

.....

**THANK YOU FOR VOLUNTEERING TO PARTICIPATE IN THIS RESEARCH. THE FOLLOWING QUESTIONS ASK ABOUT YOUR GENERAL HEALTH, HEALTH BEHAVIOURS AND MOOD. PLEASE READ THE QUESTIONS CAREFULLY AND ANSWER TO THE BEST OF YOUR ABILITY.**

## HEALTH MATTERS. (p.2)

*Please respond to the following questions by placing a tick in the appropriate box*

In the **past year** have you suffered from a medical condition which has resulted in you being hospitalised? Yes ❑ No ❑

If yes, what was the nature of this condition?.....

.....

Do you suffer from a chronic illness Yes ❑ No ❑

If yes, what is the nature of your illness?.....

**DEPRESSION HISTORY:** (Please respond if applicable)

How long have you had depression for? .....

What treatments have you tried in the past .....

.....

Do you currently seek professional help for your depression? .....

.....

*MAJOR LIFE EVENTS*

In the last **3 months** has there been any **event** which has had **a major impact** on your life?
Yes ❑ No ❑

If yes, would you please describe the event? .....

.....

**ALCOHOL AND DRUGS**

**How often** do you normally drink ALCOHOL? everyday or most days ..... ❑
2-3 times per week ..... ❑
less than 2 times per week ..... ❑
never ..... ❑

**How much** do you normally drink in any one session? 1-2 drinks ..... ❑
3-6 drinks ..... ❑
more than 6 drinks..... ❑

Do you smoke TOBACCO? Yes ❑ No ❑

**If yes,**

On average, **how many** cigarettes **per day** do you smoke? .....

Are you currently taking any **PRESCRIBED MEDICATION**? Yes ❑ No ❑ (p.3)
**If yes**, please indicate below the type of medication you are taking and the dosage:

| **Drug** | **Dosage** |
|---|---|
| ---------- | ---------- |
| ---------- | ---------- |
| ---------- | ---------- |

Do you use **RECREATIONAL DRUGS** Yes ❑ No ❑
(for example; marijuana, speed, crack, heroin, etc.)

**If yes**,
What do you use?.....

How often would you use this/these drug(s)?
- everyday or most days ❑
- 1-2 times per week ❑
- less than once per week ❑
- less than once per month ❑

**SLEEP**
Would you describe yourself as someone who has difficulties with sleep Yes ❑ No ❑
If yes, please indicate which of the following symptoms apply to you:

- In general, would describe sleep as poor. .....❑
- Difficulty falling asleep. .....❑
- Difficulty falling asleep after waking- toss and turn, lie there worrying.....❑
- Night sweats.....❑
- Night mares.....❑
- Wake up startled during the night, feeling anxious, racing heart and short of breath. .....❑
- Wake during the night and worry. .....❑
- Wake feeling tired.....❑
- Nap often during the day.....❑
- Never seem to get enough sleep.....❑
- Tend to oversleep, but still feel tired.....❑
- Snoring.....❑
- Other (please describe).....

.....

How often do you experience these symptoms:
- Most nights ❑
- Every other night ❑
- Only occasionally ❑

## MOOD

## HOW DO YOU FEEL RIGHT NOW? (p.4)

On a scale of **1**(feeling very relaxed & calm) to **10** (feeling very anxious & uptight), please indicate, by circling one number only, how **RELAXED AND CALM** or **ANXIOUS AND UPTIGHT** you feel **RIGHT NOW**.

1 2 3 4 5 6 7 8 9 10

very relaxed & calm — very uptight & anxious

## FEELINGS & EMOTIONS

This scale consists of a number of words that describe different feelings and emotions. Read each item and then circle the appropriate answer next to the word. Indicate **to what extent you have felt this way in the last WEEK**, that is, how you feel on the average. Use the following scale to record your answers.

| 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|
| **very slightly or not at all** | **a little** | **moderately** | **quite a bit** | **extremely** |

| | | | |
|---|---|---|---|
| 1 2 3 4 5 | interested | 1 2 3 4 5 | irritable |
| 1 2 3 4 5 | distressed | 1 2 3 4 5 | alert |
| 1 2 3 4 5 | excited | 1 2 3 4 5 | ashamed |
| 1 2 3 4 5 | upset | 1 2 3 4 5 | inspired |
| 1 2 3 4 5 | strong | 1 2 3 4 5 | nervous |
| 1 2 3 4 5 | guilty | 1 2 3 4 5 | determined |
| 1 2 3 4 5 | scared | 1 2 3 4 5 | attentive |
| 1 2 3 4 5 | hostile | 1 2 3 4 5 | jittery |
| 1 2 3 4 5 | enthusiastic | 1 2 3 4 5 | active |
| 1 2 3 4 5 | proud | 1 2 3 4 5 | afraid |

For Women Only: (p.5)

Because feelings and emotions are affected by different stages of the menstrual cycle would you please indicate what stage of your cycle you are at:

- ❑ First week (menstruation)..... *Day1-7*
- ❑ Second week..... *Day 8-14*
- ❑ Third week..... *Day 15-21*
- ❑ Fourth week..... *Day22 to end of cycle*
- ❑ No longer have periods.

What is the average length (in days eg.21, 28, or 32) of your menstrual cycle? .....

**If you have any questions, please don't hesitate to ask.**

**THANK YOU FOR YOUR COOPERATION AND PARTICIPATION.**

NOTES:.....

.....

.....

.....

.....

.....

.....

# APPENDIX G

Study 2 Sleep Diary.

Name: .....  SLEEP DIARY  Date: .....

| | **Example** Tuesday | **Day 1** | **Day 2** | **Day 3** | **Day 4** | **Day 5** | **Day 6** | **Day 7** |
|---|---|---|---|---|---|---|---|---|
| **Lights out** | 11:45 pm | | | | | | | |
| **Mins to fall asleep** | 20 | | | | | | | |
| **Waking times & (mins awake)** | 1:50 (10 mins) 3:45 (90 mins) | | | | | | | |
| **Final waking time** | 7:30am | | | | | | | |
| **Reasons for waking (on each occasion)** | 1. Nightmare 2. Outside noise | | | | | | | |
| ***Feeling on waking (Rate 1 to 10)** | 4 | | | | | | | |
| **Total Sleep time** | 5hrs 45min | | | | | | | |
| **General Comments From participant and or partner** | Very restless, muscle jerks in the night | | | | | | | |
| **Medications eg. sleeping tablets** | Nil | | | | | | | |
| **No. of cups of tea or coffee after 6pm** | 3 | | | | | | | |

*Rate your 'Feeling on Waking' from 1 2 3 4 5 6 7 8 9 10

(the worst I have ever felt) (the best I have ever felt)

# APPENDIX H

Study 2 Light and Mood Diary.

**Name:**..... **LIGHT THERAPY & MOOD DIARY** **DATE** .....

Please use this diary to record the time of day you use your lightmask and then rate your feeling before and again after using the mask.

| | **Example** | **Day 1** | **Day 2** | **Day 3** | **Day 4** | **Day 5** | **Day 6** | **Day 7** |
|---|---|---|---|---|---|---|---|---|
| **Week 1** | Tue 22/3 | | | | | | | |
| **Time of day** | 9pm | | | | | | | |
| **Rate feeling before** | 5 | | | | | | | |
| **Rate feeling after** | 6 | | | | | | | |
| **Comments** | unsettled | | | | | | | |
| **Week 2** | | | | | | | | |
| **Time of day** | 9.10pm | | | | | | | |
| **Rate feeling before** | 7 | | | | | | | |
| **Rate feeling after** | 6 | | | | | | | |
| **Comments** | relaxed | | | | | | | |
| **Week 3** | | | | | | | | |
| **Time of day** | 9.15pm | | | | | | | |
| **Rate feeling before** | 6 | | | | | | | |
| **Rate feeling after** | 6 | | | | | | | |
| **Comments** | OK | | | | | | | |
| **Week 4** | | | | | | | | |
| **Time of day** | 9pm | | | | | | | |
| **Rate feeling before** | 7 | | | | | | | |
| **Rate feeling after** | 8 | | | | | | | |
| **Comments** | good | | | | | | | |

*Rate your Feeling **before** and **after** using the lightmask from 1 2 3 4 5 6 7 8 9 10

(the worst I have ever felt) (the best I have ever felt)

# APPENDIX I

Lightmask™ manufacture's information sheet.

# The LightMask

The LightMask is a low-cost device for flickering light therapy (photic stimulation), based on the research into PMS and migraine conducted by Dr. Duncan Anderson at the Royal Postgraduate Medical School at Hammersmith Hospital in London.

Dr. Anderson's flickering light treatment has been developed into a small portable device suitable for home use. The LightMask looks somewhat like a conventional eye shade. It has a soft cotton outer cover and weighs less than two ounces, making it comfortable to wear. There are no wires or switches and the LightMask is turned on by pressing a concealed button. After fifteen minutes it switches off automatically, allowing it to be used when going to sleep. Inside the LightMask a high-speed microprocessor and associated software implement the Anderson Protocol, the flickering light sequences developed by Dr. Anderson. The LightMask is powered by built-in batteries. The batteries do not need to be replaced, but every few weeks they need to be recharged by plugging the LightMask into a wall transformer.

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LightMask CE

# APPENDIX J

Study 2, 'Participant Information Sheet'.

INFORMATION SHEET FOR PARTICIPANTS.
THE USE OF PHOTIC STIMULATION TO TREAT MOOD AND SLEEP PROBLEMS.

This research aims to assess the value of photic stimulation (or light therapy) in the treatment of mood and sleep disorder. Photic stimulation involves the delivery of a flashing light stimulus of a particular frequency, through closed eyelids, using a mask with small lights on the inner surface of each eye cover. The purpose of this is to produce changes in the electrical activity of the brain. Research has shown that particular frequencies of light can be conducive to the production of positive mood states and assist in improving the quality of sleep.

Photic stimulation is considered to be problem free for individuals who do not have **epilepsy,** or **migraine** and it is requested that you don't participate if you have a history of either of these conditions. As a further precaution you will not be permitted to join the study if you have **bipolar disorder** or **psychosis**. Because certain medications affect the EEG (electroencephalography) we regret we will not be able to admit you to the study if you are currently taking **medication for depression or anxiety**.

**The study runs over a 3month period and includes the following three phases:**

**1. Initial phase**: The initial assessment will include a 2hour session where you will be given information about the study and you can ask questions you have about the study and the people conducting it, and we will answer your questions to the best of our ability. We will give you details about the measurements taken, the equipment used, what is required of you during the study and your rights as a participant. You will then be asked to sign a consent form before starting the study. During this session baseline measures will be taken of your brainwave activity or EEG. You will also be asked to fill out questionnaires related to your mood, sleep habits, psychological functioning, and lifestyle factors such as drinking and smoking habits. In addition information and instructions about the treatment schedule and the sleep diary requirements will be given to you.

**2. Intervention phase**: The intervention phase will begin one week after the initial assessment. During this **4 week period** you will be using a 'light mask' once per day for about 20minutes and keeping a diary of your mood and sleep patterns. If you are in the wait list group you will simply be keeping a sleep diary for the month. During this month measures of your brainwave activity and depression level will be taken at week 2 and again at week 4. Then there will be a 2month break before the final follow-up.

**3. Follow-up phase:** At the end of the experiment, 2 months after using the light mask and/or keeping a sleep diary, final measures of your sleep patterns (sleep diary for 1week), brainwave activity (EEG), mood and psychological functioning will be done. At this time you will be fully debriefed regarding the experimental condition you were in and the intended effects of the light stimulation you received.

**Your rights as a participant:**

- You have the right to withdraw from this study at any time.
- You have the right to ask questions about the study, and that procedures be fully explained.
- You have the right to expect that the researchers involved will behave in a professional and ethical manner and at no time knowingly put you at risk.

If you have any questions regarding this study please ring Kerry Leahan on 6244 2648, or Carmel O'Sullivan on 6125 6536, at the Division of Psychology, The Australian National University, during office hours or you can contact Kerry after hours on 0419 281 698. ANU protocol number: 2000/104/ ACTCC Ethics ETH3/01.114. Date 6/7/2001

# APPENDIX K

Means and standard deviations for study 2 variables.

Appendix K: Means and standard deviations (SD) for Study 2 variables: for waitlist (WL), and photic stimulation (PS) groups, continuous light (CL), 22Hz, 5Hz, & 13Hz.

**Mean (SD) log alpha power ($\mu V^2/Hz$) across sessions (alpha asymmetry)**

| group | site | baseline | session2 | session3 | follow-up |
|---|---|---|---|---|---|
| **WL** | F3 | 1.66 (0.79) | 1.50 (0.88) | 1.68 (0.76) | 1.64 (0.99) |
| (n=10) | F4 | 1.61 (0.81) | 1.49 (0.92) | 1.63 (0.75) | 1.67 (0.89) |
| **CL** | F3 | 1.22 (0.81) | 1.34 (0.76) | 1.38 (0.84) | 1.43 (0.91) |
| (n=10) | F4 | 1.17 (0.78) | 1.27 (0.75) | 1.23 (0.87) | 1.31 (0.94) |
| **22Hz** | F3 | 1.27 (0.86) | 1.28 (0.87) | 1.31 (0.80) | 1.44 (1.03) |
| (n=9) | F4 | 1.23 (0.89) | 1.23 (0.92) | 1.28 (0.85) | 1.34 (1.04) |
| **5Hz** | F3 | 1.80 (0.66) | 1.92 (0.56) | 1.85 (0.62) | 2.00 (0.70) |
| (n=11) | F4 | 1.79 (0.67) | 1.90 (0.56) | 1.87 (0.64) | 2.01 (0.70) |
| **13Hz** | F3 | 1.49 (0.76) | 1.66 (0.72) | 1.63 (0.74) | 1.61 (0.71) |
| (n=11) | F4 | 1.40 (0.71) | 1.59 (0.71) | 1.52 (0.73) | 1.49 (0.67) |

Mean (SD) 10Hz EEG magnitude for eyes closed and 10Hz PS across EEG sites.

| eyes closed | | | | | |
|---|---|---|---|---|---|
| **group** | **site** | **baseline** | **session2** | **session3** | **follow-up** |
| **WL** | Fp1 | 0.77 (0.37) | 0.69 (0.47) | 0.77 (0.42) | 0.79 (0.47) |
| (n=10) | Fp2 | 0.75 (0.38) | 0.68 (0.50) | 0.75 (0.42) | 0.79 (0.45) |
| | F3 | 0.95 (0.40) | 0.86 (0.49) | 0.96 (0.41) | 0.92 (0.55) |
| | F4 | 0.91 (0.42) | 0.85 (0.54) | 0.93 (0.41) | 0.94 (0.51) |
| | C3 | 1.09 (0.47) | 1.00 (0.55) | 1.10 (0.41) | 1.10 (0.60) |
| | C4 | 1.06 (0.46) | 1.02 (0.61) | 1.11 (0.44) | 1.09 (0.59) |
| | P3 | 1.39 (0.64) | 1.32 (0.68) | 1.38 (0.63) | 1.45 (0.73) |
| | P4 | 1.42 (0.58) | 1.37 (0.66) | 1.42 (0.57) | 1.43 (0.71) |
| | O1 | 1.57 (0.81) | 1.60 (0.83) | 1.60 (0.70) | 1.70 (0.92) |
| | O2 | 1.54 (0.74) | 1.54 (0.79) | 1.49 (0.67) | 1.68 (0.84) |
| **CL** | Fp1 | 0.06 (0.50) | 0.65 (0.49) | 0.65 (0.53) | 0.69 (0.58) |
| (n=10) | Fp2 | 0.56 (0.51) | 0.62 (0.48 | 0.60 (0.55) | 0.68 (0.59) |
| | F3 | 0.82 (0.49) | 0.85 (0.52) | 0.85 (0.54) | 0.93 (0.58) |
| | F4 | 0.78 (0.52) | 0.83 (0.51) | 0.78 (0.57) | 0.87 (0.62) |
| | C3 | 1.10 (0.52) | 1.05 (0.46) | 1.03 (0.48) | 1.14 (0.56) |
| | C4 | 0.94 (0.52) | 0.98 (0.51) | 0.92 (0.56) | 1.06 (0.58) |
| | P3 | 1.33 (0.73) | 1.42 (0.59) | 1.38 (0.64) | 1.48 (0.67) |
| | P4 | 1.04 (0.72) | 1.46 (0.62) | 1.38 (0.64) | 1.47 (0.65) |
| | O1 | 1.68 (0.85) | 1.75 (0.64) | 1.71 (0.69) | 1.79 (0.72) |
| | O2 | 1.66 (0.81) | 1.72 (0.66) | 1.73 (0.70) | 1.76 (0.65) |
| **22Hz** | Fp1 | 0.67 (0.55) | 0.67 (0.51) | 0.68 (0.42) | 0.80 (0.60) |
| (n=9) | Fp2 | 0.65 (0.53) | 0.66 (0.49) | 0.67 (0.43) | 0.78 (0.61) |
| | F3 | 0.78 (0.51) | 0.78 (0.53) | .078 (0.43) | 0.86 (0.61) |
| | F4 | 0.76 (0.53) | 0.76 (0.55) | 0.76 (0.48) | 0.83 (0.62) |
| | C3 | 0.85 (0.50) | 0.88 (0.55) | 0.87 (0.44) | 0.89 (0.63) |
| | C4 | 0.88 (0.50) | 0.90 (0.55) | 0.85 (0.47) | 0.85 (0.60) |
| | P3 | 1.22 (0.77) | 1.20 (0.78) | 1.13 (0.64) | 1.13 (0.86) |
| | P4 | 1.25 (0.66) | 1.25 (0.73) | 1.18 (0.60) | 1.23 (0.79) |
| | O1 | 1.59 (0.93) | 1.47 (0.88) | 1.42 (0.80) | 1.42 (1.02) |
| | O2 | 1.57 (0.81) | 1.47 (0.84) | 1.40 (0.78) | 1.45 (0.89) |

Appendix K: Means and standard deviations for Study 2 variables, continued.

| eyes closed continued: | | | | | |
|---|---|---|---|---|---|
| **group** | **site** | **baseline** | **session2** | **session3** | **follow-up** |
| **5Hz** (n=11) | Fp1 | 1.00 (0.50) | 1.08 (0.47) | 1.07 (0.40) | 1.10 (0.47) |
| | Fp2 | 1.01 (0.48) | 1.09 (0.45) | 1.05 (0.40) | 1.09 (0.46) |
| | F3 | 1.15 (0.47) | 1.21 (0.40) | 1.19 (0.44) | 1.20 (0.44) |
| | F4 | 1.18 (0.46) | 1.22 (0.42) | 1.22 (0.45) | 1.25 (0.44) |
| | C3 | 1.34 (0.45) | 1.38 (0.43) | 1.37 (0.53) | 1.38 (0.44) |
| | C4 | 1.41 (0.46) | 1.45 (0.40) | 1.47 (0.51) | 1.47 (0.48) |
| | P3 | 1.84 (0.57) | 1.96 (0.61) | 1.87 (0.67) | 1.97 (0.58) |
| | P4 | 1.95 (0.56) | 2.06 (0.62) | 1.99 (0.67) | 2.04 (0.56) |
| | O1 | 2.24 (0.65) | 2.35 (0.68) | 2.21 (0.67) | 2.30 (0.63) |
| | O2 | 2.19 (0.60) | 2.30 (0.70) | 2.19 (0.65) | 2.27 (0.61) |
| **13Hz** (n=11) | Fp1 | 0.74 (0.39) | 0.84 (0.41) | 0.78 (0.41) | 0.78 (0.41) |
| | Fp2 | 0.70 (0.39) | 0.82 (0.39) | 0.77 (0.41) | 0.77 (0.38) |
| | F3 | 0.93 (0.45) | 1.04 (0.45) | 1.00 (0.44) | 0.99 (0.41) |
| | F4 | 0.88 (0.44) | 1.00 (0.45) | 0.95 (0.43) | 0.94 (0.41) |
| | C3 | 1.13 (0.54) | 1.25 (0.58) | 1.18 (0.54) | 1.18 (0.48) |
| | C4 | 1.12 (0.56) | 1.24 (0.59) | 1.17 (0.57) | 1.15 (0.53) |
| | P3 | 1.38 (0.60) | 1.51 (0.66) | 1.46 (0.60) | 1.44 (0.51) |
| | P4 | 1.45 (0.65) | 1.59 (0.67) | 1.57 (0.66) | 1.52 (0.59) |
| | O1 | 1.61 (0.57) | 1.71 (0.64) | 1.76 (0.56) | 1.61 (0.50) |
| | O2 | 1.71 (0.53) | 1.78 (0.61) | 1.86 (0.59) | 1.73 (0.54) |
| photic stimulation | | | | | |
| **WL** (n=10) | Fp1 | 1.03 (0.30) | 1.06 (0.28) | 0.99 (0.29) | 0.88 (0.35) |
| | Fp2 | 1.01 (0.31) | 1.03 (0.28) | 0.98 (0.29) | 0.85 (0.34) |
| | F3 | 1.13 (0.34) | 1.26 (0.30) | 1.12 (0.31) | 1.00 (0.44) |
| | F4 | 1.10 (0.37) | 1.22 (0.33) | 1.12 (0.29) | 1.04 (0.35) |
| | C3 | 1.21 (0.43) | 1.35 (0.36) | 1.23 (0.32) | 1.21 (0.47) |
| | C4 | 1.24 (0.42) | 1.32 (0.36) | 1.26 (0.29) | 1.17 (0.43) |
| | P3 | 1.50 (0.55) | 1.58 (0.51) | 1.60 (0.43) | 1.55 (0.60) |
| | P4 | 1.58 (0.47) | 1.61 (0.42) | 1.64 (0.41) | 1.57 (0.54) |
| | O1 | 1.90 (0.41) | 2.05 (0.44) | 1.99 (0.36) | 1.96 (0.51) |
| | O2 | 1.89 (0.37) | 1.94 (0.38) | 1.94 (0.39) | 1.96 (0.50) |
| **CL** (n=10) | Fp1 | 1.01 (0.44) | 1.05 (0.44) | 1.09 (0.48) | 1.11 (0.50) |
| | Fp2 | 0.98 (0.42) | 1.05 (0.40) | 1.08 (0.44) | 1.13 (0.48) |
| | F3 | 1.13 (0.48) | 1.18 (0.49) | 1.24 (0.50) | 1.26 (0.52) |
| | F4 | 1.14 (0.41) | 1.21 (0.44) | 1.22 (0.49) | 1.24 (0.51) |
| | C3 | 1.26 (0.38) | 1.27 (0.35) | 1.39 (0.34) | 1.39 (0.34) |
| | C4 | 1.21 (0.32) | 1.27 (0.30) | 1.34 (0.34) | 1.34 (0.37) |
| | P3 | 1.72 (0.44) | 1.69 (0.44) | 1.78 (0.38) | 1.73 (0.47) |
| | P4 | 1.69 (0.43) | 1.77 (0.39) | 1.78 (0.38) | 1.73 (0.44) |
| | O1 | 2.19 (0.54) | 2.21 (0.53) | 2.24 (0.50) | 2.23 (0.51) |
| | O2 | 2.14 (0.46) | 2.13 (0.49) | 2.21 (0.44) | 2.17 (0.42) |
| **22Hz** (n=9) | Fp1 | 0.93 (0.37) | 0.92 (0.42) | 1.04 (0.42) | 0.97 (0.35) |
| | Fp2 | 0.90 (0.39) | 0.90 (0.42) | 1.02 (0.43) | 0.97 (0.36) |
| | F3 | 1.10 (0.35) | 1.11 (0.45) | 1.19 (0.42) | 1.13 (0.31) |
| | F4 | 1.08 (0.37) | 1.09 (0.47) | 1.18 (0.42) | 1.10 (0.34) |
| | C3 | 1.17 (0.35) | 1.18 (0.45) | 1.23 (0.40) | 1.20 (0.33) |
| | C4 | 1.17 (0.37) | 1.18 (0.48) | 1.25 (0.39) | 1.20 (0.33) |
| | P3 | 1.49 (0.56) | 1.49 (0.60) | 1.45 (0.58) | 1.44 (0.51) |
| | P4 | 1.56 (0.45) | 1.51 (0.61) | 1.50 (0.53) | 1.54 (0.41) |
| | O1 | 2.07 (0.54) | 2.03 (0.54) | 2.05 (0.44) | 1.96 (0.48) |
| | O2 | 2.13 (0.42) | 2.07 (0.54) | 2.09 (0.52) | 2.01 (0.34) |

Appendix K continued: Means and standard deviations for Study 2 variables.

| photic stimulation continued: | | | | | |
|---|---|---|---|---|---|
| **group** | **site EC** | **baseline** | **session2** | **session3** | **follow-up** |
| **5Hz** (n=11) | Fp1 | 1.09 (0.19) | 1.20 (0.28) | 1.21 (0.21) | 1.29 (0.26) |
| | Fp2 | 1.08 (0.20) | 1.16 (0.29) | 1.19 (0.21) | 1.28 (0.23) |
| | F3 | 1.15 (0.18) | 1.28 (0.28) | 1.28 (0.21) | 1.35 (0.22) |
| | F4 | 1.18 (0.20) | 1.30 (0.30) | 1.33 (0.24) | 1.38 (0.21) |
| | C3 | 1.41 (0.31) | 1.50 (0.37) | 1.51 (0.33) | 1.48 (0.36) |
| | C4 | 1.44 (0.36) | 1.61 (0.32) | 1.57 (0.27) | 1.60 (0.39) |
| | P3 | 2.04 (0.45) | 2.13 (0.49) | 2.09 (0.57) | 2.19 (0.39) |
| | P4 | 2.13 (0.49) | 2.29 (0.45) | 2.19 (0.54) | 2.29 (0.42) |
| | O1 | 2.49 (0.51) | 2.56 (0.41) | 2.51 (0.50) | 2.61 (0.33) |
| | O2 | 2.45 (0.54) | 2.59 (0.40) | 2.54 (0.48) | 2.62 (0.35) |
| **13Hz** (n=11) | Fp1 | 0.85 (.047) | 0.97 (0.43) | 0.91 (0.43) | 0.95 (0.47) |
| | Fp2 | 0.85 (0.44) | 0.95 (0.42) | 0.95 (0.43) | 0.92 (0.47) |
| | F3 | 1.05 (0.51) | 1.13 (0.42) | 1.11 (0.43) | 1.17 (0.45) |
| | F4 | 1.04 (0.49) | 1.13 (0.36) | 1.11 (0.39) | 1.17 (0.41) |
| | C3 | 1.28 (0.50) | 1.33 (0.44) | 1.30 (0.42) | 1.35 (0.46) |
| | C4 | 1.35 (0.51) | 1.39 (0.40) | 1.37 (0.42) | 1.38 (0.46) |
| | P3 | 1.60 (0.52) | 1.75 (0.47) | 1.70 (0.42) | 1.65 (0.45) |
| | P4 | 1.75 (0.63) | 1.87 (0.51) | 1.81 (0.52) | 1.78 (0.51) |
| | O1 | 2.01 (0.56) | 2.05 (0.57) | 2.11 (0.51) | 1.97 (0.60) |
| | O2 | 2.01 (0.61) | 2.10 (0.58) | 2.15 (0.58) | 2.03 (0.60) |

**Mean (SD) rated well-being before and after PS** (range 1=worst to 10= best).

| **group** | **time** | **week1** | **week 2** | **week 3** | **week 4** |
|---|---|---|---|---|---|
| **CL** (n=10) | before | 4.32 (1.42) | 4.59 (1.19) | 4.95 (1.45) | 4.78 (1.45) |
| | after | 5.04 (1.04) | 5.06 (1.21) | 5.40 (1.55) | 5.27 (1.52) |
| **22Hz** (n=10) | before | 5.21 (.71) | 5.37 9.76) | 5.37 (.48) | 5.48 (.67) |
| | after | 5.87 (.84) | 6.00 (1.26) | 6.06 (.76) ( | 6.13 (.90) |
| **5Hz** (n=11) | before | 5.18 (1.15) | 5.02 (.95) | 5.46 (1.14) | 5.54 (1.33) |
| | after | 5.48 (1.33) | 5.64 (1.26) | 6.07 (1.59) | 6.09 (1.68) |
| **13Hz** (n=9) | before | 4.96 (.86) | 5.36 (.71) | 5.44 (.82) | 5.44 (1.20) |
| | after | 5.16 (.97) | 5.56 (.83) | 5.64 (.87) | 5.57 (1.26) |

**Mean (SD) subjective relaxation across sessions**
(range: 1= very uptight & anxious to 10 very relaxed & calm)

| **group** | **baseline** | **session 2** | **session 3** | **follow-up** |
|---|---|---|---|---|
| **WL** (n=10) | 6.40 (2.27) | 6.00 (1.89) | 6.40 (1.65) | 6.80 (1.55) |
| **CL** (n=10) | 7.10 (1.73) | 5.70 (1.89) | 6.90 (1.52) | 5.90 (2.02) |
| **22Hz** (n=10) | 5.60 (2.22) | 5.20 (1.75) | 6.00 (1.25) | 5.60 (2.17) |
| **5Hz** (n=11) | 6.73 (2.45) | 6.45 (1.75) | 7.09 (2.17) | 6.27 (1.62) |
| **13Hz** (n=11) | 5.55 (1.51) | 6.45 (1.57) | 6.09 (1.81) | 6.91 (1.64) |

Appendix K continued: Means and standard deviations for Study 2 variables.

**Mean (SD) positive affect (PANAS) across sessions** (range: 10-50).

| group | baseline | session 2 | session 3 | follow-up |
|---|---|---|---|---|
| **WL** (n=10) | 25.50 (10.54) | 26.90 (7.96) | 29.70 (8.18) | 30.00 (10.98) |
| **CL** (n=9) | 21.33 (8.80) | 28.11 (8.37) | 26.33 (6.12) | 27.44 (8.37) |
| **22Hz** (n=9) | 25.33 (6.20) | 27.55 (5.41) | 27.33 (4.97) | 27.77 (6.65) |
| **5Hz** (n=12) | 29.33 (8.20) | 28.83 (7.67) | 31.42 (6.21) | 29.16 (7.92) |
| **13Hz** (n=11) | 24.64 (7.42) | 28.64 (5.77) | 25.91 (6.93) | 30.18 (7.08) |

**Mean (SD) negative affect (PANAS) across sessions** (range: 10-50).

| group | baseline | session 2 | session 3 | follow-up |
|---|---|---|---|---|
| **WL** (n=10) | 26.40 (7.66) | 22.80 (7.84) | 21.80 (5.61) | 20.30 (7.48) |
| **CL** (n=10) | 26.20 (9.39) | 20.10 (8.16) | 16.50 (8.63) | 18.20 (5.95) |
| **22Hz** (n=10) | 26.20 (7.74) | 23.40 (11.11) | 21.60 (9.75) | 23.60 (9.22) |
| **5Hz** (n=12) | 31.66 (8.01) | 25.67 (7.44) | 21.17 (8.63) | 23.67 (8.80) |
| **13Hz** (n=11) | 24.18 (8.31) | 20.00 (6.21) | 19.82 (4.04) | 20.36 (8.16) |

**Mean (SD) depression levels (BDI-II) across sessions** (range: 0-63).

| group | baseline | session 2 | session 3 | follow-up |
|---|---|---|---|---|
| **WL** (n=10) | 22.88 (11.14) | 19.11 (7.41) | 14.33 (7.62) | 15.11 (12.10) |
| **CL** (n=10) | 26.50 (13.01) | 11.00 (9.14) | 12.50 (9.63) | 15.00 (11.20) |
| **22Hz** (n=10) | 24.50 (12.03) | 14.00 (9.51) | 14.40 (13.66) | 14.60 (11.24) |
| **5Hz** (n=11) | 20.17 (10.47) | 13.08 (9.89) | 8.42 (7.77) | 13.75 (10.63) |
| **13Hz** (n=9) | 24.90 (10.22) | 14.64 (7.28) | 17.09 (11.06) | 12.45 (8.81) |

**Mean (SD) 'sleep onset latency' in minutes for six weeks of sleep diary.**

| group | baseline | week 1 | week 2 | week 3 | week 4 | follow-up |
|---|---|---|---|---|---|---|
| **WL** (n=10) | 21.57 (13.05) | 30.26 (25.70) | 19.20 (13.49) | 19.24 (15.10) | 15.88 (8.38) | 18.20 (10.60) |
| **CL** (n=10) | 44.73 (37.20) | 32.34 (21.38) | 34.10 (28.11) | 25.04 (27.68) | 30.25 (34.59) | 20.76 (15.90) |
| **22Hz** (n=10) | 42.07 (70.57) | 46.44 (89.46) | 40.71 (71.41) | 33.74 (50.05) | 45.42 (94.30) | 47.07 (77.91) |
| **5Hz** (n=12) | 46.81 (72.59) | 24.83 (21.10) | 29.86 (25.28) | 24.31 (22.57) | 23.17 (28.85) | 41.60 (58.30) |
| **13Hz** (n=10) | 20.80 (14.67) | 23.43 (15.39) | 22.07 (12.80) | 18.53 (12.85) | 17.40 (15.96) | 16.02 (8.70) |

Appendix K: Means and standard deviations for Study 2 variables, continued.

**Mean (SD) number of night wakings for six weeks of sleep diary.**

| group | baseline | week 1 | week 2 | week 3 | week 4 | follow-up |
|---|---|---|---|---|---|---|
| **WL** (n=10) | 1.69 (2.45) | 1.35 (1.11) | 1.05 (0.81) | 1.38 (0.97) | 1.55 (1.48) | 0.86 (0.75) |
| **CL** (n=10) | 1.39 (0.84) | 1.27 (1.16) | 0.93 (1.05) | 1.00 (1.00) | 0.94 (0.96) | 0.92 (0.88) |
| **22Hz** (n=10) | 1.68 (1.12) | 1.16 (0.85) | 1.09 (0.88) | 1.05 (0.84) | 1.01 (1.14) | 0.84 (0.73) |
| **5Hz** (n=12) | 1.64 (1.02) | 1.62 (1.03) | 1.19 (0.96) | 104 (0.86) | 1.36 (0.95) | 1.36 (0.98) |
| **13Hz** (n=10) | 1.30 (0.84) | 1.20 (1.01) | 1.19 (0.96) | 1.00 (8.86) | 1.02 (0.83) | 1.07 (0.72) |

**Mean (SD) 'sleep efficiency' (percentage) across six weeks of sleep diary.**

| group | baseline | week 1 | week 2 | week 3 | week 4 | follow-up |
|---|---|---|---|---|---|---|
| **WL** (n=10) | 91.17 (4.12) | 88.72 (6.53) | 91.45 (4.53) | 91.35 (5.68) | 93.19 (2.59) | 93.28 (4.25) |
| **CL** (n=10) | 82.43 (11.96) | 87.25 (8.18) | 87.95 (9.21) | 87.01 (12.41) | 87.72 (10.48) | 90.71 (7.06) |
| **22Hz** (n=10) | 81.88 (17.24) | 84.87 (18.05) | 86.42 (17.67) | 86.06 (15.33) | 84.51 (20.97) | 87.25 (16.11) |
| **5Hz** (n=12) | 78.88 (15.20) | 81.92 (12.70) | 84.57 (10.92) | 84.79 (10.15) | 85.52 (13.05) | 81.77 (16.86) |
| **13Hz** (n=10) | 87.58 (9.55) | 91.29 (5.96) | 90.07 (8.66) | 90.71 (7.45) | 90.22 (7.95) | 90.01 (8.00) |

**Mean (SD) 'waking mood' across six weeks of sleep diary.**

| group | baseline | week 1 | week 2 | week 3 | week 4 | follow-up |
|---|---|---|---|---|---|---|
| **WL** (n=10) | 3.99 (0.73) | 4.59 (1.03) | 4.65 (1.05) | 4.88 (1.11) | 4.68 (0.98) | 5.12 (1.35) |
| **CL** (n=10) | 3.98 (0.65) | 4.20 (1.11) | 4.14 (1.13) | 4.47 (1.13) | 4.46 (1.26) | 4.65 (1.10) |
| **22Hz** (n=10) | 4.18 (1.11) | 5.01 (0.91) | 5.45 (0.68) | 5.38 (1.09) | 5.57 (1.49) | 5.82 (0.83) |
| **5Hz** (n=12) | 4.40 (1.40) | 4.93 (1.17) | 5.13 (1.24) | 5.23 (1.29) | 5.58 (1.80) | 5.10 (1.48) |
| **13Hz** (n=10) | 4.48 (0.57) | 4.92 (0.88) | 5.11 (0.78) | 5.25 (0.89) | 5.15 (1.10) | 5.31 (0.88) |

# APPENDIX L

Ranges for Pearson's correlation coefficients ranges for Study 2 variables.

Appendix L: Ranges for Pearson's correlation coefficients across sessions for study 2 variables across sessions.

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 global theta | 1.0 | | | | | | | | | | | | | |
| 2 global alpha | [.71**, .77**] | 1.0 | | | | | | | | | | | | |
| 3 global beta | [.60**, .63**] | [.77**, .79**] | 1.0 | | | | | | | | | | | |
| 4 PS response | [-.01, .18] | [-.05, .14] | [-.13, .10] | 1.0 | | | | | | | | | | |
| 5 subjective relaxation | [-.17, .04] | [-.37*, .03] | [-.27*, .09] | [-.30*, .10] | 1.0 | | | | | | | | | |
| 6 depression | [-.19, .02] | [-.19, .05] | [-.13, .05] | [-.12, .05] | [-.55**, -.34*] | 1.0 | | | | | | | | |
| 7 anxiety | [.01, .01] | [-.19, -.11] | [-.14, -.08] | [.03, .11] | [-.41**, -.34**] | [.51**,.66**] | 1.0 | | | | | | | |
| 8 global severity index | [-.13, -.01] | [-.20, -.14] | [-.22, -.08] | [-.08, .06] | [-.45**, -.37**] | [.69**, .76**] | [.83**, .84**] | 1.0 | | | | | | |
| 9 positive affect | [-.03, .16] | [.03, .17] | [-.04, .18] | [-.01, .08] | [.16, .50**] | [-.59**, -.34**] | [-.22, -.14] | [-.39**,-.30*] | 1.0 | | | | | |
| 10 negative affect | [-.12, .15] | [-.09, .09] | [-.06, .03] | [-.12, .13] | [-.46**, -.27*] | [.41**, .61**] | [.58**,.69**] | [.50**,.55**] | [-.24,.14] | 1.0 | | | | |
| 11 sleep onset | [-.08, -.01] | [-.15, -.02] | [-.09, .13] | [-.01, .34*] | [-.25, .01] | [-.10, .32*] | [.13, .17] | [.15, .19] | [-.31*, .02] | [-.08, .21] | 1.0 | | | |
| 12 number of wakings | [.12, .26] | [.16, .31*] | [.15, .30*] | [-.19, .15] | [-.02, .11] | [-.28*, .04] | [-.34**, -.03] | [-.38**, -.10] | [.04, .22] | [-.11, .15] | [-.21, -.05] | 1.0 | | |
| 13 sleep efficiency | [-.07, .07] | [-.13, -.01] | [-.20, -.06] | [-.30*, -.11] | [-.11, .25] | [-.21, .16] | [-.11, -.07] | [-.07, -.05] | [.14, .29*] | [-.12, .13] | [-.85**,-.78**] | [-.10, .06] | 1.0 | |
| 14 alpha asymmetry | [-.08, .07] | [-.16, .01] | [.03, .07] | [-.04, .17] | [-.11, -.02] | [.07, .14] | [.23, .26] | [.16, .26] | [-.08, .12] | [.02, .27*] | [.05, .29*] | [-.19, .11] | [-.29*,-.05] | 1.0 |
| 15 menstrual cycle (n=23) | [.10] | [.28] | [.17] | [.05] | [-.19] | [-.17] | [-.17] | [-.19] | [.08] | [-.01] | [.24] | [-.18] | [-.03] | [.15] |

* significance LE . 05 ** significance LE . 01